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RENAL FUNCTIONAL TESTS FROM THE BIOCHEMICAL VIEW POINT.[1]

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THE PHYSIOLOGY OF RENAL SECRETION.

Histological Anatomy of the Kidney.

THE essential histological structure of the kidney consists of an invaginated capsule (Bowman's capsule) which leads by a narrow neck into a long tubule. The tubule soon after leaving the capsule becomes convoluted (first convoluted tubule) and then proceeds to form a long U-shaped loop (loop of Henle) which passes from the cortex of the kidney to the medulla and then doubles back again to become convoluted again in the vicinity of the Bowman's capsule (second convoluted tubule) and then proceeds to a collecting tubule. The latter is joined by several fellows and opens through a calyx on to the pelvis of the kidney. The cells lining different portions of this unit show characteristic differences, being flattened in the capsule, high and striated in the convolutions, pavement-like in the descending limb of the loop and columnar in the loop and ascending limb. The invagination of the capsule is caused by a tuft of capillary blood vessels (the glomular tuft), the afferent vessel of which comes direct from branches of the renal artery. Reflected over each capillary of the tuft, like the fingers of the glove follow the hand,(1) is the epithelium of Bowman's capsule. The capillaries of the tuft collect into an efferent arteriole which is of somewhat smaller calibre than the afferent vessel. This efferent vessel then breaks up into a further series of capillaries which are distributed

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 31, 1930.

to the tubules. From these capillaries emerge veins which join up and ultimately form the renal vein (see Figure I).

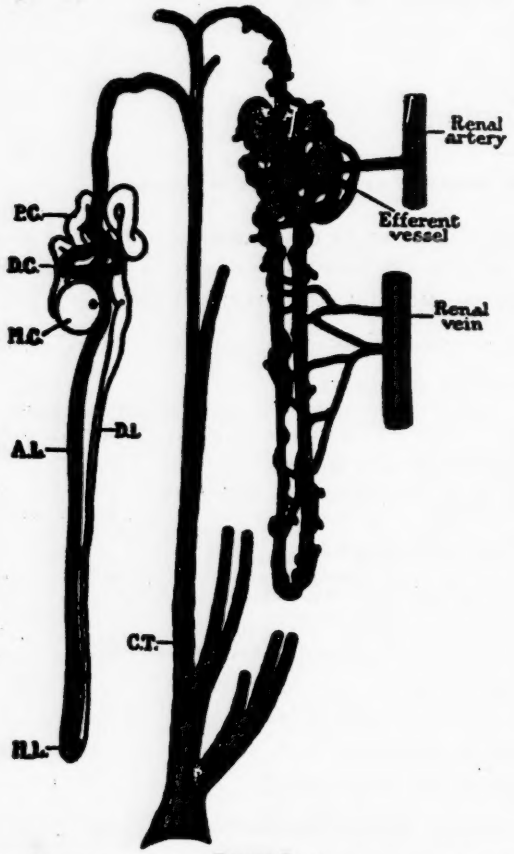


FIGURE I.

After Cushny. On the left the tubule is drawn after a diagram of G. C. Huber. The tubule is outlined from the capsule to the loop of Henle, and is shaded from that point to the end of the collecting tubule. On the right a diagram of the circulation is added: M.C. = Malpighian corpuscle; P.C. = proximal convoluted tubule; D.L. = descending limb; H.L. = loop of Henle; A.L. = ascending limb; D.C. = distal convoluted tubule; C.T. = collecting tubule.

There are various calculations of the number of glomeruli in a human kidney. Thus Rehberg[2] gives the figure of 1,000,000, while Traut[3] considers the number approaches 4,500,000. In either case the area available for the passage of fluid from the capillaries into the capsule is exceedingly great.

The Work Done in Secreting Urine.

The concentration of electrolytes in the urine is much greater than that of the blood and a large amount of work has to be done to accomplish this. The secretion of one litre of urine requires the consumption of one litre of oxygen, which is equivalent to five calories,[4] or in other words, one litre of urine requires about one-eleventh of the energy an average man produces in an hour under basal conditions. The energy is obtained partly from the blood pressure, but mostly from cell activity.

Theories of Renal Secretion.

How, then, is this energy expended? There is as yet no satisfactory theory of renal secretion. Briefly the main theories are: The vital theory of Bowman and Heidenhain which postulates the diffusion through the glomerulus of all the constituents of the plasma except colloids and sugar and the "vital" secretion of salts by the tubule cells. Amongst other phenomena this theory does not explain diuresis. The rival theory of Ludwig advocates in its original form that urine is a physical filtrate through Bowman's capsule of a slightly alkaline fluid containing all the constituents of the plasma except the colloids and that a reabsorption of water occurs during passage through the tubules. This theory fails to explain how various crystalloids found in the urine are concentrated to markedly varying extents over their respective concentration in the blood, for example, in one experiment the creatinine in the urine was concentrated two hundred times, whilst chloride was present to the same extent in the urine and blood.[5] "The modern theory" of Cushny[3] explains renal secretion as a mechanical filtration through Bowman's capsule of a fluid which contains all the components of plasma except the proteins, fats and colloids, and that during the passage through the tubules there is reabsorption back of from 60% to 99% of the water in the glomerular filtrate. At the same time, there is a selective reabsorption of an optimal concentration of crystalloids which are of further use to the organism. The substances which are thus reabsorbed are termed threshold bodies. Such threshold bodies are excreted only when their concentration in the blood is above their optimum or threshold. The most important of these threshold bodies are chloride, bicarbonate, sugar, amino acids and sodium. Some substances, though of some importance to the organism, are nevertheless concentrated to a considerable extent in the urine and are only partly reabsorbed by the tubule cells. These are low threshold bodies; chief among them are urea, urates and phosphates. Still other substances are quantitatively excreted and are termed "no threshold bodies." The no threshold bodies are creatinine, sulphate and foreign substances, dyes, toxins *et cetera*. Briefly this theory postulates that the function of the kidney is to restore the crystalloids of the blood to their optimal concentration by the excretion in the urine of any excess.

The kidney has also to excrete two other substances, water which is the vehicle in which all other substances are dissolved, and acid radicals which are in excess in the blood when a mixed diet is eaten. The removal of acid is partly accomplished by the quantitative excretion of sulphates and by the change of the neutral mixture of phosphates which is present in the serum, to a mixture of phosphates in which dihydrogen phosphate is in excess. According to the "modern theory," this conversion of phosphates is accomplished by the active reabsorption through the cells of the tubules of bicarbonate and sodium. The conversion of urea or

some other parent substance into ammonia is another important factor in removing acid radicals, especially in acidosis. The manner by which this conversion is accomplished is as yet only dimly guessed at.[7]

Since the formulation of Cushny's theory many serious criticisms have appeared.[6] [2] To meet certain objections Rehberg advocates the hypothesis that substances which have no threshold, but which are not actively rejected by the organism, diffuse back through the tubules, the chief examples being urea and alcohol, while substances of further use, for example, chloride, are actively reabsorbed, as postulated by Cushny.

Lastly, Continental investigators[7] have evolved a three gland theory which postulates the glomerulus as the water excretion gland, the ascending limb of Henle's loop as the salt excretion gland and the convoluted tubules as the nitrogen excretion gland.

No apology is needed for introducing theories of renal secretion into this discussion, because until the physiology, pathology and the morbid clinical states of renal diseases can be brought into a common relationship, all tests for renal efficiency will tend to remain unsatisfactory.

Renal Function Tests.

For the purpose of this paper the following scheme will be taken to represent the function of the kidney:

1. The quantitative excretion of no threshold substances, for example, creatinine, sulphate, foreign substances, dyes, poisons, toxins *et cetera*.
2. The excretion of the major portion of readily diffusible substances, for example, urea, phosphates, alcohol, diastase *et cetera*.
3. The excretion of excess of threshold substances, for example, sugar, chloride, bicarbonate, sodium, potassium *et cetera*.
4. Protector of hydrogen ion concentration of the blood, by excretion of any excess of acid or alkali radicals; formation of ammonia.
5. Preservation of water balance.

Functional tests have been evolved to test all these different phases of renal activity. The tests include examination of both blood and urine. Functional tests using no threshold substances as basis of test: Creatinine,[9] indigo-carmine, phenolsulphonephthalein[10] have been used. Functional tests have been used with low threshold substances as basis of the test: Urea has been chiefly used with various modifications, for example, the urea concentration test of Maclean and de Wesselow,[6] the urea test of Archer and Robb,[12] of Addis[13] and of Calvert.[24] Diastase estimations for many years were much used, but have now rightly fallen into disfavour.

Threshold substances have been the subject of many investigations as test substances. Amongst the most modern investigations are those with the chlorides of sodium and potassium by de Wesselow.[13] Investigators have tested renal function as a protector of hydrogen ion concentration by giving acid[14] or alkali,[15] while the ability to deal with water has received a very large amount of attention.[16] [28] Certain investigators have attempted to study the comparative value of different tests on the same subjects. By far the best attempt that I have seen was by Bowen.[29]

Test substances have been given orally, subcutaneously or intravenously and have included substances normally present in the blood, for example, urea, chlorides, sodium and potassium, creatinine *et cetera*, or of chemicals foreign to the organism, either inorganic compounds, for example, thiosulphate or organic, for example, indigo-carmine, phenolsulphonephthalein.

In renal disease in addition to changes in the concentration of normal substances between the blood and urine, abnormal products may appear; examination for these products is often of value in determining the type and extent of renal disease, for example, indican may be found in the blood[17] [18] [19] [20] while in the urine albumin, casts, blood cells, lipoid, bacteria and crystals may be present. Certain substances which are normal constituents of the blood and with whose metabolism the kidney is not directly concerned, may in certain renal disorders be present in abnormal concentration. Examples of such substances are cholesterol and calcium. The quantitative estimation of these substances has also been used for tests of renal efficiency.[21] [22] Changes in the serum colloids[23] and in water content of the plasma and the presence of anæmia have also been investigated in relation to renal function.

From this wide and expanding choice of tests some discretion must be exercised for the development in any one laboratory of a routine type of examination which will give the greatest information with the smallest amount of discomfort to the patient.

On the whole, most authorities agree that some form of urea concentration test is the most satisfactory. This should be accompanied by the examination of the fasting blood urea and complete routine urinalysis. The following has been my routine practice: The patient for examination abstains from the ingestion of much fluid for thirty-six hours previous to the test and from all food and fluid for twelve hours before the test. On the morning of the test blood obtained by venipuncture is taken into a tube containing lithium oxalate. The bladder is emptied into a specimen jar labelled "before," fifteen grammes of urea in one hundred cubic centimetres of water are administered orally and the bladder is emptied at the end of each hour for three hours into bottles labelled first hour, second hour, third hour. During the test the patient should remain quiet. The blood is examined for urea content, degree of hydræmia, if any, for creatinine content, if the urea be raised. The urine samples are each examined for percentage urea by the hypobromide method, quantity, specific gravity, reaction and presence of albumin. The specimen obtained before the administration of urea is centrifuged and the

deposit is examined for casts, red blood cells, leucocytes, refractile material, crystals *et cetera*.

The normal range of values is as follows:

Blood urea: 15 to 40 milligrammes (for normal figures of medical students in New South Wales, see Priestley and Hindmarsh[27]).

Creatinine: 0 to 1·5 milligrammes per hundred cubic centimetres.

Cholesterol (for estimation in nephrosis): 100 to 175 milligrammes per hundred cubic centimetres.

Urea concentration factor: $\frac{\text{urine urea}}{\text{blood urea}}$ 60 upwards, (that is, the kidney should be able to concentrate urea at least sixty times).

Urea concentration test: The normal values are set out in Table I.

TABLE I.

Urea Concentration Test (Normal Values).

Time.	Urea Percentage.	Specific Gravity.	Quantity in Cubic Centimetres.	Reaction.	Albumin.
Before	1·5 upwards	1010 upwards		Acid	—
1 hour	1·5 upwards	1006 upwards	Less than 150	?	—
2 hours	2·5 upwards	1010 upwards	Less than 100	?	—
3 hours	3·0 upwards	1015 upwards	Less than 80	?	—

The points to notice are: (i) That in my opinion a normal kidney of an Australian, provided there is no diuresis, during the second and third hour is capable of excreting urea above 3%. (ii) The specific gravity should rise as the available fluid for excretion becomes less in amount. (iii) There should be considerable elasticity in the output of water, preferably a sharp diuresis for the first hour and then a gradually diminished output.

As practically 100% of tests on patients in whom no definite evidence of renal disease could be discovered, complied with the conditions laid down above, I think we can confidently look with suspicion on any patient whose results fall short of the above requirements. I will point out as we proceed how several patients whose kidneys were inefficient when seen at one stage (patients examined during an attack of nephritis) were able to come up to the standards laid down when the renal condition improved, while others who were considered not to have efficient kidneys, because they did not comply with the standards and whose clinical condition has progressed, have shown further renal inefficiency at subsequent tests.

Before proceeding, I should like to stress what has been so ably said by Coope[26]:

> It is obvious that in investigating a complicated "system" like the conditions produced by nephritis in the human body, such a test as the urea concentration test is a physiological experiment in which many of the factors must necessarily be uncontrolled, and one which moreover, strictly speaking, tells us the response of the body only at the time of the test.

In giving an opinion as to the result of the renal functional test the information gleaned should be examined as a whole.

For the purposes of this paper I have classified all the tests done privately during the last five years and have also used a number of case records from the Royal Prince Alfred Hospital. I have attempted to trace all the private patients whose tests were inefficient, and am deeply indebted to the medical men who referred the patients for their interest and cooperation in letting me know the after history.

The following four groups can be separated.

Group I.

The first is a group in which there was no renal defect or in which the only renal abnormality discovered was albuminuria. This group can be divided arbitrarily according to the degree of albuminuria: Group IA had albumin – or trace, Group IB had albumin +, Group Ic had albumin more than +.

Group IA comprised by far the majority of the total number of all patients examined and to my mind it is in this group that renal efficiency tests are of the greatest importance, because by their aid one can definitely rule out any condition of chronic azotæmia nephritis.

These patients were referred for the reasons set out in Table II.

TABLE II.

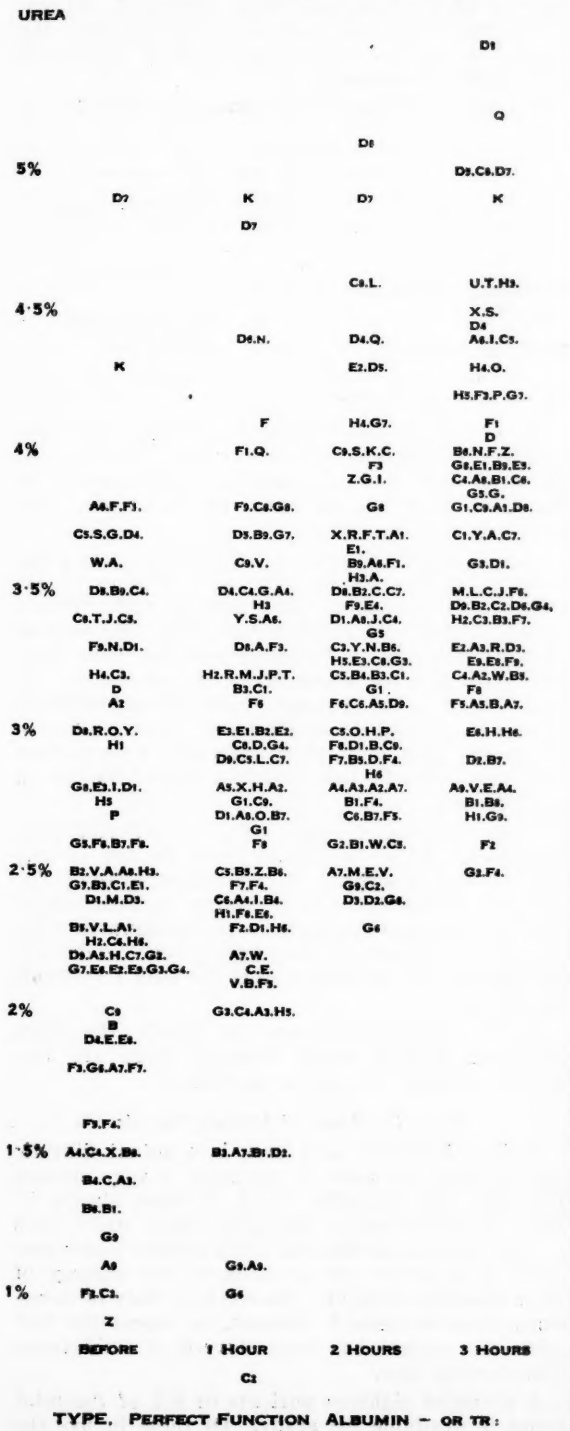
Condition.	Number of Cases.	Centrifuged Deposit.
Arteriosclerosis	2	
Arteriosclerosis and diabetes	1	
Hyperpyesis	8	Three showed red blood cells
Hyperpyesis and syncopal attacks	1	Casts present.
Œdema of legs	3	
Polyuria	2	One showed casts.
Headaches	4	
Headaches and œdema	1	
Prostatitis	3	Two showed casts.
Albuminuria	6	
Albuminuria, adolescent	7	One showed casts.
Albuminuria of pregnancy	1	
Albuminuria in previous pregnancy	6	
Albuminuria and asthma	1	
Albuminuria and paroxysmal tachycardia.	1	
Malaise	5	One showed casts.
Hæmaturia	1	
Groups referred for insurance, preoperative tests, *et cetera*.	42	Eight showed casts.
Previous history of nephrosis	1	
Pyelitis of pregnancy	1	
Extra systole	1	
Renal calculus	2	
Myxœdema	1	
Gout	2	
TOTAL	103	

The results of the tests of all these patients showed a normal blood urea, a satisfactory concentration of urea, a specific gravity of the hourly samples that answered the criterion demanded for a normal test, and the hourly quantities of urine also satisfied the criterion of normal function.

The results of the concentration of urea have been made into the form of a graph, and each patient has been given a letter so that the results of any patient can be ascertained (see Figure II, III and IV).

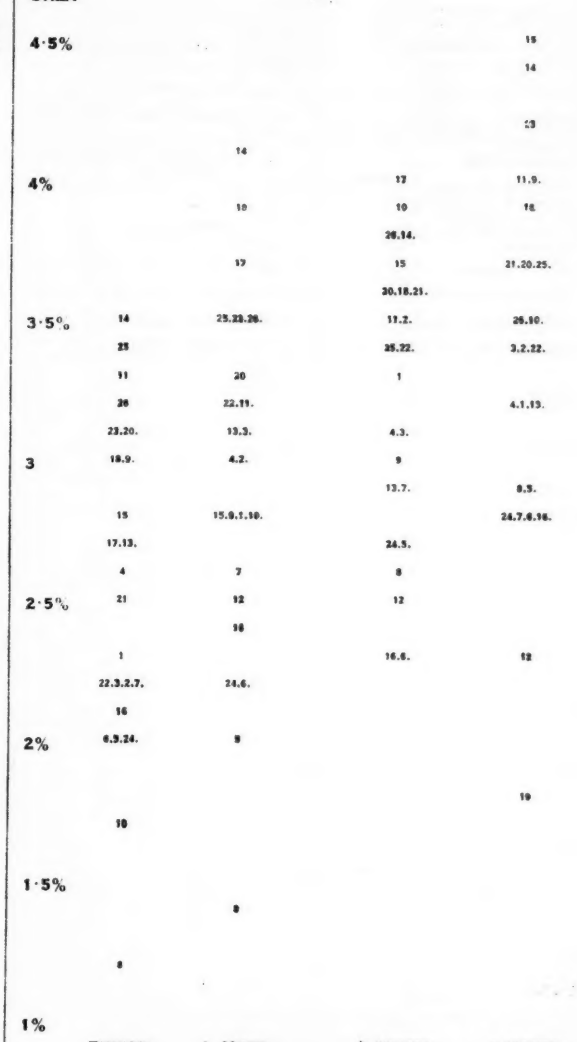
In these cases 85 out of 103, or 82%, showed the highest concentration of urea in the third hour specimen. Only twelve of these patients did not reach a concentration of 3% of urea at some stage

of the test, but all concentrated above 2·6%. Four of the twelve patients had considerable diuresis, but the test on the whole was so satisfactory that



TYPE. PERFECT FUNCTION ALBUMIN — OR TR:

CHART I.



UREA CONCENTRATION TEST

TYPE. PERFECT FUNCTION ALBUMIN + (LESS THAN +++)

CHART II.

they were considered to have quite normal function. Of the remaining eight patients, five got up to 2·8% or more and were otherwise normal in every way, leaving three patients in whom no definite damage could be proven and who must thus be considered to have unimpaired kidneys.

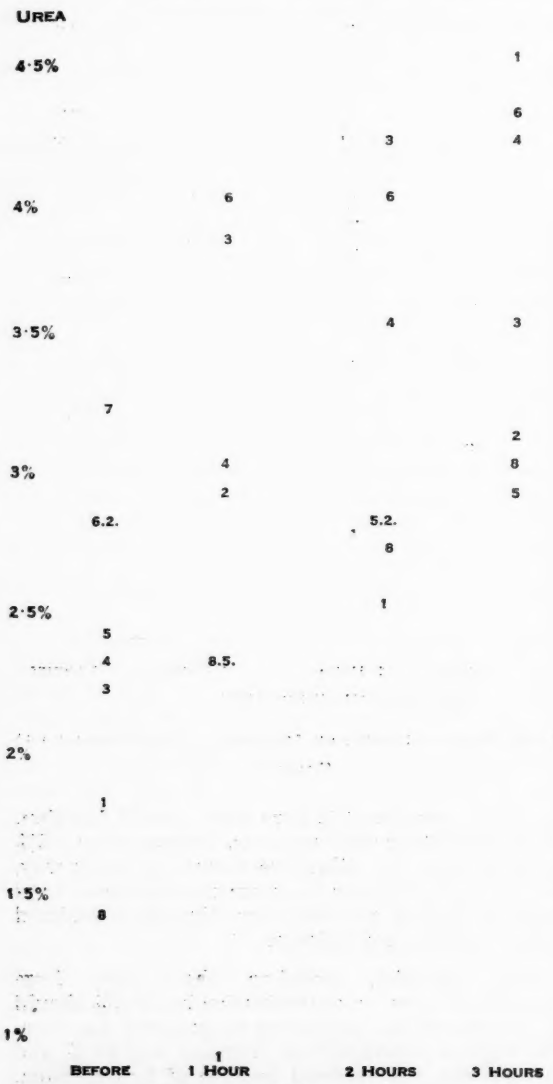
One concludes, therefore, that apart from exceptional cases no concentration below 3% should be considered as indicating unimpaired function. The highest concentration obtained was 5·6% and this patient was referred because of hypertension.

Group Ib were referred for the reasons set out in Table III.

TABLE III.

Condition.	Number of Cases.	Centrifuged Deposit.
Insurance	11	Two showed red blood cells.
History of previous nephritis or nephrosis	4	
Hydronephrosis	1	Red blood cells and whit blood cells.
Prostatitis with retention	1	
Frequency of micturition	1	
Adolescent albuminuria	3	
Arteriosclerosis and hemiplegia	1	Showed casts.
Hyperpyesis	1	
TOTAL	23	

Nineteen cases or 83% showed the maximal concentration of urea in the third hour. Seventeen of this series or 70% were able to concentrate urea to 3% or over, four concentrated to 2·9%, while the other two (one patient with hydronephrosis, one patient with prostatitis with retention) concentrated to 2·8%; these latter patients (taking the results of the tests as a whole) were considered to have efficient kidneys.



UREA CONCENTRATION TEST

TYPE. PERFECT FUNCTION ALBUMIN ++ OR +++

CHART III.

Group Ic were referred as set out in Table IV.

TABLE IV.

Condition.	Number of Cases.	Centrifuged Deposit.
Albuminuria of adolescence	3	Two showed casts.
Albuminuria of adolescence with renal calculus	1	
Albuminuria	3	Two showed casts, one white blood cells.
Recent history of nephritis	1	Numerous casts.
TOTAL	8	

All of these patients concentrated urea above 3% and five concentrated urea above 4%. Seven of the eight or 88% had the greatest concentration in the third hour. In all cases the blood urea was well within normal limits.

It is of great importance to know what the prognosis in such cases is. The after history of six patients is available. The patient with "recent nephritis" has gone steadily down hill. The patient with calculus had a lithotomy and has done well. One patient with albuminuria of adolescence is quite well. One patient with albuminuria of adolescence had tonsillectomy and albumin is present now only after exertion. One patient with albuminuria has arteriosclerosis, but is in good health.

These patients, of course, were not suffering from nephrosis. Urea functional tests do not in any way assess the severity of cases of nephrosis. The interest of Group Ic is in trying to ascertain if one is justified in ignoring gross albuminuria when it occurs as the sole abnormal symptom. No definite opinion can yet be formed from the data I was able to collect.

Before discussing cases in which the tests indicated definite renal damage, there are two groups, II and III, to be mentioned.

Group II. Cases of Marked Diuresis.

It is well known that urea is a potent diuretic and in some patients its produces a very marked diuresis. On the other hand, it must always be borne in mind that a damaged kidney which fails to concentrate metabolites, must remove waste products at a lower concentration by the passage of large amounts of urine. Therefore, it may be necessary, when diuresis is present, to repeat the test after the patient has been starved of fluid for a considerable time.

A group of eighteen patients or 8% of the total series is available for study. Of these in five the concentration of urea was 2·3% or over, the specific

gravity was satisfactory and there was neither retention of urea nor any abnormality in the centrifuged deposit, so that the renal efficiency was deemed unimpaired and a repetition was not considered necessary.

In five cases (Table V) in which the concentration of urea was unsatisfactory, the test was confirmed after further abstinence from fluid; in two the diuresis was not repeated and the concentration of urea was satisfactory, 4·6% in one case and 2·9% in the other. In three the diuresis was repeated and in all three cases there was a constancy of urea concentration, quantity per hour and specific gravity and there were casts and other abnormalities in the deposit, so that the opinion was given that the efficiency was impaired. In all these cases the blood urea was within normal limits. Nothing of value would accrue from any further consideration of the other seven cases, as I consider that their renal efficiency has not been satisfactorily ascertained.

Group III.

Some patients respond to the ingestion of urea by excreting during each of the three hours of the test a urine which is constant in amount, specific gravity

TABLE V.

Protocols of Patients with Diuresis who had Tests Repeated after Further Abstinence from Fluid.

Particulars of Patient.	Blood Urea. Milligrammes *per centum.*	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum.*	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
Male, aged 40–50	21	1·45	1012		++	Granular casts.	
		1·35	1006	210	+		
		1·75	1008	140	+		
		2·05	1010	120	+		
Repeat of test		4·10	1032		+		
		3·50	1022	90	Tr.		
		4·20	1024	50	+		
		4·60	1026	45	+		
Male. Insurance Case	26	0·4	1004		–	Nil abnormal.	
		0·6	1004	200	–		
		0·7	1004	260	–		
		0·8	1006	290	–		
Repeat of test		2·5	1016		–	A few granular casts.	
		2·4	1012	90	–		
		2·9	1014	60	–		
		2·9	1016	65	–		
Male, aged 34. Cystitis for three years		2·0			Tr.	All specimens turbid. Epithelial cells, pus cells, casts, *débris.*	
		2·3	1010	140	Tr.		
		2·2	1012	120	Tr.		
		2·4	1012	120	Tr.		
Repeat of test		1·9	1016				
		2·0	1012	105			
		2·0	1014	120			
		2·2	1014	110			
Male, aged 40. Calculi of both kidneys	25	1·1	1008		+	Numerous pus cells.	
		1·2	1010	175	+		
		1·6	1010	135	+		
		1·8	1010	125	+		
Repeat of test		1·8	1008		+		
		1·8	1016	105	+		
		1·9	1016	135	+		
		1·7	1016	80	+		
Four months later		2·1	1014		++	Pus in all specimens.	
		2·2	1014	75	++		
		2·2	1012	55	++		
		2·4	1012	55	++		
Eight months later	36 Creatinine 4·6	1·2	1010		Tr.	Pus in all specimens.	Thinks he is holding his own. Looks worse. Blood shows hydræmia.
		1·3	1010	125	Tr.		
		1·3	1010	125	Tr.		
		1·4	1010	125	Tr.		
Male, aged 70	55 Creatinine 3	1·6	1014		+++	A few granular and hyalogranular casts.	
		1·4	1010	180	++		
		1·3	1010	190	++		
		1·7	1012	90	++		
Repeat of test		2·0	1012		++		
		1·5	1010	135	++		
		1·6	1010	135	++		
		1·4	1010	140	++		
		2·1	1012	60	+++		

In urea concentration tests first line = Before.
Subsequent lines, first, second and third hour after 15 grammes of urea.

and urea percentage. This, of course, is characteristic of the damaged kidney and many examples will be seen in the protocols of patients with nephritis. Sometimes the specific gravity is high and the concentration of urea very satisfactory. Have such patients impairment of renal function? I consider that results of this nature indicate that such kidneys have no reserve; any tax on the excretory powers forces the available healthy tissue to work at its full capacity. I have seen such results in cases of destruction of one kidney and in patients suffering from focal nephritis or in whom compensatory hypertrophy following nephritis seems to have occurred. One feels that such patients should not be allowed too much strain on their kidneys and should be looked upon like patients with compensated cardiac hypertrophy. There were ten such patients in the series and the protocols of those of whom I have subsequent information are shown in Table VI.

Group IV.

We next come to consider the group of patients whose results indicated renal inefficiency—Group IV. There were 52 such patients and the after history of 42 of these is known. Nine are considered cured (see Table VII) either from the results of subsequent tests (three cases) or from the clinical opinion of their medical advisers (six cases).

One was a remarkable case:

A female, aged seven years, had complete anuria ten days after appendicectomy. The patient was semicomatose when blood was taken for analysis. The blood urea was 137 milligrammes *per centum*, creatinine was six milligrammes *per centum*.

Twelve hours after the test on Dr. Blackburn's advice double decapsulation of the kidneys was performed by Sir Alexander MacCormick. The child passed 900 cubic centimetres (thirty ounces) of urine in the next ten hours. This had a urea content of 2%, a specific gravity of 1010; it was acid and had a trace of albumin, no sugar or acetone, no casts, numerous pus cells and urates.

TABLE VI.

Protocols of Patients with Apparently Good Functional Results, but where Kidneys are apparently Working at their Fullest Capacity—The Compensated Kidney.

Particulars of Patient.	Blood Urea. Milligrammes *per centum*.	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum*.	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
Male. Insurance Case. No definite symptoms.	48	2·0	1018		–	A few casts and leucocytes.	Two and a half years later still well.
		2·1	1014	90	–		
		2·2	1014	80	–		
		2·2	1014	90	–		
Female, aged 59. Hydronephrosis with total loss of function on that side.	30	1·8	1014		–		Affected kidney removed. Patient in good health eighteen months later.
		3·3	1016	55	–		
		3·3	1016	60	–		
		3·3	1016	60	–		
Male, aged 38. Had acute nephritis at war. No symptoms now.	42	2·7	1018		–	Nil in deposit.	Quite well two and a half years later.
		2·7	1012	65	–		
		2·7	1012	60	–		
		2·9	1016	50	–		
Male, aged 40. First seen during attack of acute nephritis.	112					Albumin. Blood and casts of all types.	
One month later	47	1·9	1012		++	Blood and casts of all types.	
		2·2	1010	80	+		
		2·5	1010	65	+		
		2·5	1010	60	+		
One month later	25	1·6	1014			Casts and some red blood cells.	
		2·1	1010	70	Tr.		
		2·3	1010	70	Tr.		
		2·3	1010	70	Tr.		
One year later. (Had septic teeth out in interim.) Now on full diet.	46	2·9	1024		–	A few leucocytes.	Probably a case of focal nephritis.
		3·0	1012	86	–		
		3·3	1018	66	–		
		3·3	1020	44	–		
Male, aged 62. Prostatitis with retention.	44	1·8	1012		Tr.	Pus cells, moderate number of casts, very numerous colon bacilli.	Prostate not removed.
		2·6	1012	45	Tr.		
		2·9	1012	45	Tr.		
		3·0	1014	50	Tr.		
Male, aged 26. Left ureter obstructed by calculus. No urine on that side.	73	2·7					Died after operation.
		2·5	1020	45			
		2·5	1017	44			
		2·8	1020	42			

In urea concentration tests first line = Before.
Subsequent lines, first, second and third hour after 15 grammes of urea.

One month later the blood urea was 52 milligrammes and the creatinine was 3·5 milligrammes *per centum*. The urine contained no albumin and no casts; leucocytes and red blood cells were present. One month later the blood urea was 36 milligrammes and the creatinine 3·2 milligrammes *per centum*. The urine contained a trace of albumin as the only abnormality.

Three months later the blood urea was 35 milligrammes and the creatinine was 1·5 milligrammes *per centum*. The urine contained no abnormality.

Now after three years the girl is in perfect health and no evidence of nephritis is present from physical examination or examination of urine.

With the exception of Case VII there is adequate reason why the patients have made a good recovery. In all a successful removal of the cause of the renal defect was accomplished.

In eleven cases the condition of the patient is apparently stationary or is progressing slowly (see Table VIII).

Six patients or 13·5% have shown a definite progression (see Table IX). Finally, there were sixteen deaths (see Table X). One of these patients had malignant disease of another part of the body and the tests were performed to see if colloidal lead could be safely given. One patient, aged seventy-eight years, had arteriosclerosis.

TABLE VII.

Protocols of Patients whose Tests showed Inefficiency and who are now "Cured."

Particulars of Patient.	Blood Urea. Milligrammes *per centum*.	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum*.	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
1. Male. Nephritis and alcoholism ..	40	1·0	1002		++	Granular and hyaline casts.	Four years later doctor reports that patient gave up alcohol and is now in good health.
		1·1	1001	260	++		
		1·6	1008	130	++		
		2·5	1010	75	++		
2. Male. Acute tonsillitis and acute nephritis. Examined after attack.	38	2·1	1020		–	Pus cells, red blood cells, casts of all types.	Five years later doctor reports that patient had tonsillectomy soon after test, now in perfect health.
		2·8	1020	125	–		
		3·1	1022	75	–		
		3·1	1025	65	–		
3. Male, aged 65. Had vesical calculus removed eighteen months ago. Now had cystitis and prostatitis.	33	2·3	1014		+	Macroscopic pus in all specimens.	Prostatectomy performed. Qhite well two years later.
		2·3	1012	100	+		
		2·5	1014	35	+		
		2·5	1016	65	+		
4. Female. Had nephrolithotomy some months previously.	80	2·2	1015		–	Very few pus cells, no casts. Bacilluria.	Four years later is in very good health.
		1·8	1012	160	–		
		2·3	1015	75	–		
		2·3	1015	25	–		
5. Male, aged 80. Prostatis with retention. Nocturnal epileptiform seizures.		2·0	1011		++	Large numbers of pus cells, a few red blood cells, no casts.	
		1·9	1012	65	++		
		2·2	1012	70	++		
		2·4	1014	60	++		
After prostatectomy	49	1·1	1010		++	Pus cells and bacteria, no casts.	One year later in excellent health.
		1·3	1012	95	++		
		2·0			++		
		2·3	1012	55	++		
6. Male, aged 7. Acute nephritis. Discharge from right ear.	37	0·6	1008		+++	Blood and casts in all specimens.	
		0·7	1008	110	+++		
		1·0	1010	110	+++		
		1·3	1010	85	+++		
Had ear operated on five months later.	25	0·9	1006		–	Nil in deposit.	
		2·7	1012	110	–		
		4·0	1016	80	–		
		4·4	1018	70	–		
7. Female, aged 21. Pyelitis one year before. Headaches lately.	47	1·8	1014		++	Epithelial cells, clumps of pus cells, a few casts.	Two years later doctor reports that patient apparently quite well as she is able to play grade tennis.
		1·7	1010	55	+		
		1·6	1008	85	+		
		1·5	1010	50	+		

8. This case was described under Protocols of patients with compensated kidneys.

9. This case is described in the text.

In urea concentration tests first line = Before.
Subsequent lines, first, second and third hour after 15 grammes of urea.

TABLE VIII.

Protocols of Patients whose Condition has remained Stationary or is Progressing Slowly.

Particulars of Patient.	Blood Urea. Milligrammes *per centum.*	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum.*	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
1. Male, aged 57. Weighs 18 stone. Clinical diagnosis: Auricular fib illation, arteriosclerosis, chronic nephritis. Œdema present.	50	2·2	1018	100	++		Three and a half years later still alive and well.
		2·5	1018	65	++		
		1·8	1012	160	++		
		2·3	1012	110	++		
2. Male. Albuminuria for years. No symptoms of nephritis. One sister died of nephritis.	34	1·7	?		Tr.	No casts, numerous pus cells, colon bacilli.	
		1·7	1010	120	Tr.		
		1·7	1010	130	Tr.		
		1·7	1010	125	Tr.		
Test repeated on account of diuresis.		No specimen before.					Four years later was still quite well.
		2·1	1014	65			
		2·1	1014	65			
		2·3	1014	60			
3. Male. Frontal headaches. Systolic blood pressure, 160 millimetres of mercury.	44	1·5	1019		+	A few granular casts, a few pus cells and red blood cells.	Four years later doctor reports that patient has had cerebral hæmorrhage, but has recovered. Blood pressure still raised.
		1·9	1018	90	+		
		2·1	1018	75	+		
		2·5	1020	50	+		
4. Female, aged 40. Albumin found during seventh pregnancy. No other symptoms. Examined nine weeks *post partum.*	26	0·8	1012		++		
		1·0	1012	75	++		
		1·6	1014	70	++		
		1·7	1014	65	++		
Seen nine months later. Appeared in good health; has lot of headaches.	33	1·5	1016		++	Lot of pus and organisms—good many casts, also good deal of refractile lipoid material.	
5. Male. Arteriosclerosis, chronic nephritis, high blood pressure.	30	1·1	1018		−	Nil abnormal.	Three years later in good condition, still has high blood pressure.
		1·3	1018	70	−		
		1·5	1016	90	−		
		1·8	1016	80	−		
6. Male. General malaise. Albumin found on clinical examination.	30	1·2	1006		+++	Moderate number of casts of all types.	Six months later *in status quo.* Has recently had tonsillectomy.
		1·2	1006		+++		
		2·3	1014	70	+++		
		2·3	1016	50	+++		
7. Male, aged 30. Subacute nephritis with œdema. Blood pressure: 160 millimetres of mercury systolic and 100 diastolic.	24	1·4	1016		+++	A few granular casts and a few leucocytes.	Two years later doctor says fairly well, but still has nephritis.
		2·5	1014	110	+++		
		2·7	1018	85	+++		
		2·7	1018	70	+++		
8. Male, aged 21. Albuminuria five years. No symptoms.	69	1·4	1010		++	A few hyalogranular casts, a few leucocytes.	Four and a half years later, doctor reports that patient is still apparently quite well.[1]
		1·3	1008	115	++		
		1·3	1008	125	++		
		1·6	1008	90	++		
9. Male, aged 16. Albuminuria ten years. High blood pressure. Cardiac weakness.	32	1·8	1020		+++	A few leucocytes.	
		1·7	1014	270	+		
		3·2	1020	80	+		
		2·2	1016	100	+++		
Re-examined four years later. Patient in good health till recently, then had return of symptoms.	31	2·3	1022		+++	Moderately numerous leucocytes, a few casts, refractile lipoid material.	
		2·8	1018	150	+++		
		3·3	1020	90	+++		
		3·5	1022	70	+++		
10. Male, aged 13. Faints easily. Albumin found in urine. Systolic blood pressure, 150 millimetres of mercury.	44	1·8	1018		+++	Phosphate crystals, red blood cells, a few leucocytes, a few granular and hyalogranular casts.	Five years later very well. Systolic blood pressure, 150 millimetres of mercury. No albumin. On a liberal diet.
		2·2	1014	92	++		
		2·5	1016	135	++		
		2·7	1020	55	++		
11. Male, aged about 60. Arteriosclerosis. High blood pressure. Had hemiplegia.	59	1·5	1010		++	Moderate number of granular casts and pus cells, large number of triple phosphate crystals.	Two years later condition unchanged.
		2·0	1012	50	++		
		2·1	1010	70	++		
		2·1	1010	85	++		

In urea concentration tests first line = Before. Subsequent lines, first, second and third hour after 15 grammes of urea.

[1] This patient has been reexamined. Blood urea is 360 milligrammes, creatinine is 20 milligrammes. He is uræmic.

diabetes and gangrene and the renal lesion was a minor cause of death. One patient died from pneumonia, but, of course, the renal inefficiency probably played a determining part. The other thirteen or 30% of patients whose tests showed inefficiency and whose after history is known, died of a progressive renal disease.

An analysis of these cases shows that apart from acute nephritis, patients who have a raised blood urea content when first seen, do not have a good expectation of life. That if the blood urea is within normal limits, although the urea concentration test may yield a poor result, the prognosis is not so bad as in patients with an apparently good concentration, but who have a raised blood urea.

As Maclean[6] points out, a nephritic and a normal person on the same diet excrete approximately the same amount of urea *per diem*, but the nephritic requires a higher head of pressure. At this raised pressure the concentration of urea may be comparatively good.

When a kidney is damaged, it loses its elasticity and instead of the activity waxing and waning according to the ingestion of food and water *et*

TABLE IX.

Protocols of Patients whose Condition has Definitely Progressed.

Particulars of Patient.	Blood Urea. Milligrammes *per centum.*	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum.*	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
1. Male. Arteriosclerosis, diabetes and nephritis.	40	1·5	1030		+++	Red blood cells, moderate number of casts of all types.	Three years later doctor reports that patient has hemiplegia and is pretty bad.
		1·4	1026	150	+++		
		1·5	1026	175	+++		
		1·5	1028	150	+++		
		The high specific gravity is due to a large amount of sugar in the urine.					
2. Male, aged 50. Polyuria. Systolic blood pressure, 175 millimetres of mercury. Cardiac hypertrophy.	·27	0·7	1009		+	A few casts and pus cells.	Five years later doctor reports that blood pressure still rising and nephritic condition has progressed.
		1·0	1012	140	+		
		1·8	1012	140	++		
		2·0	1018	60	++		
3. Female, aged 50–60. Anæmia, cardiac disease and nephritis.	17	1·5	1018		−		Blood count at this time: Red cells, 4,000,000; hæmoglobin value, 36 *per centum;* colour index, 044. One year after test doctor reports that patient very breathless.
		2·2	1016	40	−		
		2·6	1018	75	−		
		2·5	1018	85	−		
4. Female, aged 22. Bilateral pyonephrosis for fifteen years. Examined soon after confinement.	47	2·1	1014		+	Numerous pus cells and bacilli, no casts.	
		2·0	1012	160	+		
		2·4	1012	80	+		
		2·3	1012	80	+		
Six months later	44	2·0	1018		++	Numerous pus cells, no casts; tubercle bacilli present.	Blood very hydræmic
		1·8	−	25	+		
		1·9	1014	65	+		
		1·9	1016	45	+		
5. Female, aged 24. Easily fatigued. Slight œdema of feet. Headaches. Systolic blood pressure, 150 millimetres of mercury.	41	1·0	1020		+++	Numerous casts of all types, much refractile lipoid material, red blood cells and leucocytes.	Four years later condition has progressed, swelling of feet more marked; uræmic symptoms a few months ago; urine unchanged.
		2·0	1014	80	+++		
		2·2	1020	40	+++		
		2·5	1020	40	+++		
6. Female, aged 36. Hyperthyreoidism, hyperpyesis, three successive abortions, persistent loss of weight. Feels perfectly well. Systolic blood pressure, 190 millimetres of mercury.	110	0·9	1010				
	Creatinine	0·9	1013	90			
	6	0·9	1015	85			
		1·2	1015	80			
		B.M.R. +34 *per centum.*					
One year later. Systolic blood pressure, 200 millimetres of mercury. Feet swell at times, feels very well; has wasted without losing weight.	Blood Urea	1·0					
	180	0·9	1008	180			
	Creatinine	0·9	1008	170			
	7	1·2	1008	35			
		B.M.R. +27 *per centum.*					

In urea concentration tests first line = Before.
Subsequent lines, first, second and third hour after 15 grammes of urea.

TABLE X.

Protocols of Patients who have Died.

Particulars of Patient.	Blood Urea. Milligrammes *per centum.*	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum.*	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
1. Male, aged 35. Infection of renal trace for fifteen years. Uræmic symptoms for last few weeks.	80 Creatinine 4·2	0·8 0·8 0·8 0·8	1004 1006 1004 1006	 100 130 90	++ ++ ++ ++	Large amount of pus and organisms, no casts, no tubercle bacilli.	Death some months later from uræmia. *Post mortem*, left kidney a mere cyst. Right kidney showed well-marked tubercle bacilli and nephritis.
2. Female. Headaches, retinal hæmorrhage.	71	1·6 1·6 1·8 2·9	1012 1010 1010 1010	 105 120 25	+++ +++ +++ +++	Definite amount of pus and red blood cells, no casts.	Died suddenly three weeks later.
3. Male, aged 60. Pains in back, œdema, general failure of health.	65 Creatinine 3	1·3 1·5 1·5 1·4	1022 1019 1017 1017	 65 100 90	+++ +++ +++ +++	Numerous casts and red blood cells.	Death six months later.
4. Male, aged 65. Prostatitis and retention of recent onset.	460 Creatinine 11	1·3 1·5 1·1 0·9	1008 1010	 195 35 20	+ + + +		Death two days later.
5. Male, aged 21. Acute nephritis aged fifteen years. Gradual failure of health since. Did not seek advice till aged twenty-one years. At that time œdema of feet and eyes; headaches.	32	2·0 2·1 2·2 2·2	1018 1016 1014 1014	 85 90 60	+++ +++ +++ +++	Large number of casts of all types, red blood cells, leucocytes, refractile and lipoid material.	
One year later	46	1·8 2·1 2·4 2·7	1018 1018 1018 1018	 80 60 60	+++ +++ +++ +++	Deposit as above.	Death from uræmia nine months later.
6. Female, aged 25. Had nephrolithotomy eighteen months ago. Now œdematous.	81 Creatinine 3·6					Pus (++), numer-colon bacilli, no tubercle bacilli.	Leucocytes, 19,000.
Three months later. Systolic blood pressure, 220 millimetres of mercury; diastolic pressure, 135 millimetres.	188 Creatinine 10	0·8	1006			A few leucocytes and casts.	
Two weeks later	250 Creatinine 15						Death in uræmia a few days later. *Post mortem* examination: Right kidney showed congenital cysts, left kidney sclerosis with areas of regeneration.
7. Male, aged 40. Dyspnœa, headaches, loss of weight. No urinary symptoms.	58	1·7 2·0 2·0 1·8	1020 1018 1015 1018	 85 85 65	++ ++ ++ ++	A few degenerate casts.	Death from uræmia within six months.
8. Male. Tertiary syphilis, hepatitis, nephritis.	37	2·0 1·4 No specimen 2·4	1010 1012 1014	 200 95			Death eighteen months later. Nephritis probably determining factor.
9. Male. Symptoms of nephritis for one year.	67	1·5 [illegible] 1·9 2·0	1016 1014 1014 1014	 190 60 20	+ + + +	Large number of casts, red blood cells and leucocytes.	Death from nephritis within six months.
10. Female. Old lady with nephritis and congenital syphilis.	41	1·5 2·0 3·0 3·2	1018 1016 1016 1018	 70 40 40	++ + + +	Numerous casts of all types, including waxy.	Death from cerebral hæmorrhage within two years.

TABLE X—(*Continued*).

Particulars of Patient.	Blood Urea. Milligrammes *per centum*.	Urea Concentration Test.				Centrifuged Deposit.	After-history.
		Urea, *per centum*.	Specific Gravity.	Quantity in Cubic Centimetres.	Albumin.		
11. Female, aged 30–40. Hyperpyesis, albumen found during pregnancy. Child stillborn at seven months.	50	1·3	1010		++	Granular casts, red blood cells and leucocytes.	Death within three years.
		1·3	1006	140	+		
		1·5	1010	100	+		
		1·6	1010	85	+		
12. Male, aged 13. History of acute nephritis and arteriosclerosis.	70	0·9	1014		+++	Moderate number of casts, numerous red blood cells and leucocytes.	
	Creatinine 2·5	0·7	1010	140	+++		
		1·0	1012	110	+++		
		1·2	1012	70	+++		
Three months later.	92	1·0	1010		+++	Very numerous casts of all types, numerous red blood cells and leucocytes.	Red cells, 3,500,000 hæmoglobin value, 50 *per centum*; colour index, 0·7.
	Creatinine 1·6	1·0	1010	90	+++		
		1·3	1008	85	+++		
		1·4	1010	50	+++		
Three months later. Systolic blood pressure, 200 millimetres of mercury.	150					Numerous casts of all types, not many red blood cells or leucocytes.	Death within one year.
	Creatinine 3·2						

In urea concentration tests first line = Before.
Subsequent lines, first, second and third hour after 15 grammes of urea.

cetera during the day, the portion of the kidney that is still functioning, is forced to work at its fullest capacity hour in, hour out. Hence the constancy of specific gravity and of water and urea output noted in the urea concentration test in nephritics. Working on the assumption of this constancy indicating a kidney working full out, I think one is justified in suspecting disease whenever this constancy is met with, no matter how high the concentration of urea or specific gravity.

Polycystic Disease.

The question of renal efficiency in polycystic disease is of importance. An analysis of seven cases confirmed by operation in the Royal Prince Alfred Hospital in which data are available, shows that six patients were able to concentrate a urine of 1016 or over, that all had a blood urea above normal or on the higher side of normality and that of five urea concentration tests only one reached a concentration of 2·5% in any hour. All patients were above twenty years of age.

In a familial group analysed by Fuller[25] there were nine examined; five were over thirty years of age, four were below twenty-one years of age. Of those above thirty, none concentrated up to 2·5% and only two above 2%. Of the four who were twenty-one or under, all concentrated above 2·5%. The lowest maximum concentration shown was 2·8%.

From these figures it seems as if some evidence of renal failure becomes apparent after the twentieth year, though the extent varies considerably, as it may not be incompatible with a long life, one of Fuller's patients being seventy-four years old and still a healthy man. The specific gravity of the urine is not necessarily low, though often stated to be so. It only becomes low when a chronic nephritic condition has developed.

Acute Nephritis.

An analysis of fifteen cases of acute nephritis from the records of the Royal Prince Alfred Hospital shows great variability in results, as would be expected, seeing that acute nephritis is such a variable disease as regards severity, duration, amount of kidney affected *et cetera*. The results of tests may vary rapidly from day to day and a very different picture can be obtained within a short time. For example, in one case the results of three successive tests at ten days' interval were as shown in Table XI.

TABLE XI.

Blood Urea.	Creatinine.	Highest Specific Gravity.	Greatest Concentration of Urea.
138 milligrammes *per centum*	7	1017	1·6 *per centum*
42 milligrammes *per centum*	1	1018	1·5 *per centum*
33 milligrammes *per centum*		1027	1·9 *per centum*

On the whole, there is always some retention of nitrogenous products and generally some loss of ability to concentrate urea. The urinary changes, of course, are characteristic, albumin, red blood cells and casts being almost invariably present. The specific gravity is very variable, ranging from 1006 to 1038 in the hospital series. The greatest value of renal function tests in acute nephritis would appear to be to test the efficiency of the kidney at subsequent periods after the acute attack and I would suggest three months, six months and a year as suitable periods. Perhaps one should stress the fact that the disease must be considered to be active so long as red blood cells are present in the urinary deposit. The problem of renal functional tests in renal conditions complicating pregnancy is broadly similar to that of acute nephritis.

Extrarenal Conditions.

There remain to be considered the changes of renal function due to extrarenal conditions which affect renal activity. Chief amongst such conditions are disorders of the cardio-vascular system. In essential hypertension or hyperpyesis the renal functional tests are often excellent and urea may be concentrated more than in health. In arteriosclerosis the kidneys may also escape serious damage and their excretory power may be unimpaired. On the other hand, both hyperpyesis and arteriosclerosis may be and often are accompanied by renal damage as evidenced by functional tests.

In cardiac disease too the kidney tests may be perfectly normal. In anæmia, not secondary to nephritis, the ability of the kidney to excrete nitrogenous products may be unimpaired. But it must be remembered that nearly all nephritic patients become anæmic. The cause of this anæmia is unknown.[30] Lastly, in any acute intoxication the functional activity of the kidneys may become impaired like that of all other organs.

There are certain tests apart from the routine ones which are of value for patients under treatment with a curtailed intake of protein. In such cases the level of the blood urea can be made to fall within normal limits, so that we must turn to some substance whose metabolism is totally endogenous, for example, creatinine, to determine the presence of nitrogen retention. The normal concentration of creatinine in the blood is from 0 to 1·5 milligrammes *per centum* and as it is readily excreted by the kidney in health, its accumulation in the blood stream is in chronic cases of very serious import. It is almost universally agreed that a concentration above six milligrammes *per centum* foretells a fatal termination within a few months. Only two patients in my experience, one with a creatinine of six milligrammes *per centum*, another with a creatinine of fifteen milligrammes *per centum*, are alive more than a year after the test.

Uric acid is practically of no value in renal functional tests because the metabolism of the purins is mainly extrarenal and changes in the level of uric acid in the blood are mostly due to extrarenal disease.

The examination for cholesterol in urinary diseases is a difficult problem. Like uric acid, its metabolism is chiefly extrarenal and its variations in individuals not suffering from any renal disorder is very great, especially in certain pituitary disorders and in anomalous fat metabolism, as in cholesteræmia, diabetes *et cetera*. Certain workers have found it useful in nephritis, especially nephritis associated with œdema, but I feel that it is always an index of extrarenal disorder and does not measure renal efficiency, though, of course, in nephrosis which I consider is a general tissue disease,[22] it may bear some apparent relation to the renal condition.

The determination of total non-protein nitrogen has no value over the determination of urea and creatinine. Urea always forms the greatest amount of this nitrogen, so that no further information is derived from doing both examinations.

The problem of renal function in health and disease is full of difficulties; our tests of function are at best comparative, so that at present a standardized technique is a first essential and the patient suspected of renal damage should be investigated in a routine manner. Advance in knowledge is unlikely where investigation is haphazard and change of method frequent. There is one further essential for the elucidation of the problem—a close cooperation between clinician, pathologist and physiologist.

REFERENCES.

(1) Vimtrup (1926): Physiology paper dedicated to Professor A. Krogh, 357, Copenhagen, quoted by P. B. Rehberg, *loco citato*.

(2) P. B. Rehberg: "Studies on Kidney Function," *The Biochemical Journal*, Volume XX, 1926, page 448.

(3) A. R. Cushny: "The Secretion of the Urine," Second Edition, page 5.

(4) David Burns: "The Introduction to Biophysics," Second Edition, page 199.

(5) P. B. Rehberg: "Studies on Kidney Function," *The Biochemical Journal*, Volume XXX, 1926, page 461.

(6) H. Maclean: "Modern Methods in the Diagnosis and Treatment of Renal Disease."

(7) Stanley Benedict and T. B. Nash: "The Origin of Ammonia in Urine," *Journal of Biological Chemistry*, Volume LXXXII, June, 1929, page 673.

(8) Pütter: "*Die Dreidrüsentheorie der Harnbereitung*," 1926.

(9) V. C. Myers: "Practical Chemical Analysis of the Blood," Second Edition, page 65.

(10) J. T. Geraghty and L. G. Rowntree: "The Phenolsulphonephthalein Test for Estimating Renal Function," *Journal of the American Medical Association*, Volume LVII, 1911, page 811.

(11) H. E. Archer and G. D. Robb: "The Tolerance of the Body for Urea in Health and Disease," *The Quarterly Journal of Medicine*, Volume XVIII, April, 1925, page 274.

(12) T. Addis: "Renal Function and the Amount of Functioning Tissue," *Archives of Internal Medicine*, September 30, 1922, page 37.

(13) O. L. V. de Wesselow: "The Excretion of Chlorides by the Healthy and Diseased Kidney," *The Quarterly Journal of Medicine*, Volume 19, October, 1925, page 53.

(14) G. C. Linder: "The Effect of Mineral Acid on Acid-base Regulation in Health and Disease," *The Quarterly Journal of Medicine*, Volume XX, April, 1927, page 285.

(15) M. Rosenberg and H. Hellfors: "*Nierenfunctionsprüfung mittels Alkalibelastung und Azidose der Nierenkranken*," *Deutsche Medizinische Wochenschrift*, July, 1929, page 1212.

(16) Leathes: *The Lancet*, Volume II, 1920, page 933.

(17) C. H. Andrewes: "An Unexplained Diazo Colour Reaction in Uræmic Sera," *The Lancet*, March 22, 1924, page 590.

(18) G. V. Rudd: "The Blood in Nephritis and Uræmia," THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1928, page 715.

(19) G. A. Harrison and R. J. Bromfield: "The Cause for the Andrewes' Diazo Test for Renal Inefficiency," *The Biochemical Journal*, January, 1928, page 43.

(20) A. Bolliger and M. S. S. Earlam: "The Andrewes Diazo Reaction," THE MEDICAL JOURNAL OF AUSTRALIA, April 12, 1930.

(21) James Maxwell: "The Blood Cholesterol in Nephritis," *The Quarterly Journal of Medicine*, Volume XXI, January, 1928, page 297 (a full list of references given at end of article).

(22) O. L. V. de Wesselow: "On the Phosphorus and Calcium in the Blood in Renal Disease," *The Quarterly Journal of Medicine*, Volume XVI, July, 1923, page 341.

(23) E. B. Mayrs: "The Functional Pathology of Nephritis," *The Quarterly Journal of Medicine*, Volume XIX, April, 1926, page 273.
(24) E. G. B. Calvert: "Diagnosis and Prognosis in Chronic Renal Disease," *The British Medical Journal*, January 10, 1925, page 64.
(25) C. J. Fuller: "Familial Polycystic Disease of the Kidneys," *The Quarterly Journal of Medicine*, Volume XXII, July, 1929, page 567.
(26) R. Coope and W. H. Jones: "The Urea Concentration Test in Nephritis and its Prognostic Value," *The British Medical Journal*, January 8, 1927, page 51.
(27) H. Priestley and Ellen M. Hindmarsh: "The Blood Urea and Nitrogen Output of Australian Students," THE MEDICAL JOURNAL OF AUSTRALIA, March 8, 1924.
(28) Albarran: "*Annales des Maladies d'Organes Genito-urinaires*," quoted by Cushny.
(29) B. D. Bowen: "A Study of the Comparative Value of Tests of Renal Function in a Group of Cases of Mild or Suspected Nephritis," *American Journal of the Medical Sciences*, Volume CLXXIV, December, 1927, page 769.
(30) "Anæmia in Bright's Disease" (Editorial), *Journal of the American Medical Association*, Volume XCIII, December, 1929, page 1812.

THE CLINICAL ASPECT OF TESTS OF RENAL EFFICIENCY.[1]

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I HAVE to confess myself guilty of having suggested the subject of renal efficiency tests as suitable for this evening's combined meeting, but little thought at that time that I should be asked to represent the Section of Medicine on an occasion so important, hoping rather to sit meekly and imbibe information, not endeavour to impart it.

From the viewpoint of the physician, tests of renal efficiency might be expected in theory, at least, to be of service:

1. To determine whether or not the function of the kidney be impaired, or whether an individual be the subject of renal disease.
2. To arrive at some conclusion regarding the actual pathological condition of the kidneys in a patient suffering from renal disease.
3. As a guide to prognosis and expectation of life in nephritic patients.
4. As an indication for appropriate treatment.
5. As a guide to the desirability of terminating gestation in patients suffering from renal disease of pregnancy.
6. To determine whether or not a patient suffering from renal disease should be considered as a reasonable risk for surgical interference, for example, in prostate disease.

Before discussing in detail the significance of the various tests which have been devised for estimating renal function, certain general principles seem to call for consideration. First let the conditions be considered in the instance of an individual otherwise normal, from whom one kidney has been removed surgically for traumatic rupture, gunshot wound or other injury.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 31, 1930.

As renal tissue, in common with that of other human organs, is provided in the individual on a scale to meet maximal demands, it is found that the patient with one sound kidney remaining will give normal response to the various tests commonly employed, although deprived of one-half of his normal renal tissue. Any gross departure from normal findings therefore must be interpreted as indicating a very considerable impairment of secretory efficiency.

It has, moreover, been authoritatively stated by Addis[(1)] that the activity of 50% of renal tissue is sufficient to remove nitrogenous waste at normal rate. Positive findings of renal inefficiency are, therefore, to be regarded as evidence of gross failure of excretory function, whereas negative findings cannot be accepted as excluding the presence of renal damage of considerable degree.

The tests of renal function commonly employed to gain information in the several conditions previously specified, may briefly be summarized:

1. Tests for the presence of abnormal urinary constituents—protein, casts *et cetera*.
2. Observations of altered physiological balance between blood and urine, as estimated by blood analysis and urine analysis; estimations of blood urea and non-protein nitrogen, cholesterol, chlorides, uric acid, creatinine *et cetera*.
3. Estimation of the eliminating power of the kidney following the administration of various substances: (i) water, (ii) urea, (iii) renal test meals, (iv) foreign substances such as dyes, phenolsulphonephthalein, indigo-carmine *et cetera*.

A brief review may be made of the information gained by these several methods.

ABNORMAL CHARACTERS AND CONSTITUENTS OF URINE.

The information gained by observation of abnormal constituents of urine, while providing information as to structural damage to the renal tissue, does not necessarily permit of accurate deduction as to the measure of impairment of secretory function.

ALTERED PHYSIOLOGICAL BALANCE BETWEEN BLOOD AND URINE.

The tests of Group II blood urea estimation, blood creatinine, blood cholesterol *et cetera*, have been used extensively and give valuable information of the excretory activity of the renal tissues at the particular time for those particular substances and have a definite sphere of usefulness in diagnosis and prognosis. Apart from renal disease, however, there are conditions which may cause an increase in nitrogenous retention in the blood, for example, prolonged vomiting, intestinal obstruction *et cetera*. The amount of urine excreted and the urea content of the urine must, therefore, be taken into consideration at the same time.

It is a generally accepted standard that a constant finding of a non-protein nitrogen content of the blood of forty milligrammes and upwards per hundred cubic centimetres, is definite evidence of renal inefficiency. It is, however, unwise to draw

deductions from a single observation of this nature, because it is well recognized that a transient ureteric or uretheral obstruction may give rise to great increase in blood urea and non-protein nitrogen apart from actual kidney disease.

A persistent increase in these readings, in the absence of obstruction or other complicating factors, is to be regarded as evidence of serious renal defect. The well known urea concentration test of Maclean[2] is of considerable value in determining the power of the kidney to deal with urea, particularly if the volume of urine voided in successive specimens be noted, so that the actual amount of urea eliminated, as well as the percentage excreted in the various samples of urine, is observed. There does not appear to be adequate evidence, however, that the ability of the kidney to excrete urea is necessarily an adequate measure of the total function of the kidney to excrete all waste products.

An attempt has been made recently by Lashmet and Newburgh[3] to devise a simple means of testing clinically the ability of the kidneys to excrete solids in solution or, in other words, to make urine. The solids excreted include not only urea, but all the substances normally excreted by the kidney. The aim of the test is to estimate the total excretory capacity, not the ability to excrete urea only or water only. The principle is to provide a definite diet, with a known amount of fluid and solids, and to estimate the function of the kidney working at a maximal capacity in a known period. The details of this method are considered further under the next heading, as the method is really analogous to a renal test meal.

Tests of Elimination of Various Substances.

Water Concentration Test.

As the water concentration test of Volhard and Koranyi[4] does not seem to be generally known, the details are briefly set out.

Technique.

The patient is instructed to take as little fluid as possible from lunch time onwards, but to eat his ordinary meal. No fluids at all are taken after 9 p.m., when bladder is emptied and urine discarded. All urine passed from this hour onwards until 9 a.m. is saved and measured. At 9 a.m. 900 cubic centimetres (a pint and a half) of weak tea or water are given and urine passed every hour for at least four and, if possible, six hours. Each hourly specimen is measured and a sample saved. The specimens are investigated as regards volume and specific gravity. The patient is kept in bed during the test and the observation is carried out more than once.

Results.

1. A normal patient should not excrete more than twenty ounces of night urine and the specific gravity should not be less than 1025. (The average volume in health is usually given as 450 cubic centimetres or fifteen ounces.)

2. Water test or dilution urine. All the 900 cubic centimetres (thirty ounces) taken in the morning should be excreted in four and a half to five hours, more than half being normally secreted in the first two hours. The maximum flow is usually in the second hour, when the volume should reach 300 to 450 cubic centimetres (ten to fifteen ounces).

3. The specific gravity should fall to 1003 or lower.

Conclusions.

A poor concentration of night urine is generally accepted as one of the earliest signs of renal impairment, the volume being increased particularly in cases of chronic nephritis of inflammatory origin, in which the specific gravity of the voided urine is usually low.

In arteriosclerotic kidney the volume of night urine is found to be slightly increased and specific gravity less reduced.

As disease progresses in both these groups of cases, the urinary output may be diminished and specific gravity tend to become fixed 1010 to 1012.

Apart from indicating the presence of disease, the test is a useful guide to the extent of renal damage of more certain prognostic value than isolated observation of high non-protein nitrogen content of the serum, which at this stage may not show gross evidence of renal defect. The method possesses the added advantage that it can readily be carried out clinically without special apparatus.

Administration of Urea: Urea Concentration Tests.

The administration of urea, followed by observations of urinary excretion, has been used extensively as a test of renal function. The test depends on flooding the blood with urea, thus raising its concentration above normal value and observing the ability of the kidney to remove the excess. The technique of the test according to the standard of Maclean, usually accepted in this country, is so well known as to render description unnecessary. The test appears, however, to be open to the objection that the result is to some extent dependent upon the rate of absorption of the test dose from the alimentary tract, which introduces a factor of uncertainty. Moreover, the possible effect of urea as a diuretic has to be taken into account, so that it would seem that the actual amount of urine voided in the several specimens should be noted as well as the urea percentage in considering the ability of the kidney to excrete the urea in a known time. Futhermore, it is to be questioned whether the ability to concentrate and to excrete urea is a true measure of the power of the kidney to excrete all the solid constituents of urine. It is, of course, undoubted that the ability to concentrate urea under the condition of the test gives important clinical information.

Renal Test Meals.

The principle of renal test meals is to give a standard diet and observe the urine excreted with regard to volume, specific gravity, urea and salt

concentration. The work of Mosenthal[5] in this connexion is well known, stress being laid upon: (i) Variations in specific gravity, (ii) balance between intake and output of salts and nitrogen, (iii) high specific gravity of night urine.

Constant findings in these observations indicate a normal response, whereas in the nephritic patient renal impairment is said to be indicated by: (i) Fixed or low specific gravity, (ii) lowered output of salts and nitrogen, (iii) polyuria, (iv) loss of concentration of night urine.

In the recent publication of Lashmet and Newburgh, already referred to, an endeavour has been made to arrange a test which provides a standard waste to be removed by the kidney during a time when it is forced to function at its maximal capacity. A standard diet is enforced for three days prior to the test, during which time the patient remains at rest in bed. The diet consists of: Protein, 45 grammes, fat 106 grammes, carbohydrate 180 grammes, total caloric value 2,000. Sodium chloride intake is two to three grammes daily.

A diet of such a nature is given as follows:

	Grammes.
Breakfast.	
Fruit (15%)	100
Cornflakes	20
Bread	60
Butter	15
Cream (20%) with coffee	100
Sugar	15
Dinner.	
Potato	100
Asparagus or tomato (stewed)	100
Lettuce salad	100
Pineapple (canned)	100
Milk	200
Bread	30
Cream	40
Supper.	
Egg (1)	
Potato (baked)	100
Spinach (cooked)	100
Green salad	100
Butter	20
Bread	30
Peaches	100
Cream (with coffee)	60

In addition, a glass of water may be taken with each meal and a glass between each meal.

At 6 p.m. on the third day after supper, all intake, both fluid and solid, is stopped until noon the following day. The urine is collected at 10 a.m. on this day and discarded. A specimen is collected at 12 noon. The specific gravity of this specimen is then accurately determined, correction being made for the variation in gravity caused by albumin content.

According to these observations, the urine so secreted by normal individuals should attain a specific gravity of 1026, a failure to concentrate urine to this figure indicating an impairment of renal function. It is claimed that by this method earlier information of renal damage may be gained than by the estimation of non-protein nitrogen in blood or by the dye tests in common use.

Dye Tests.

The agents commonly employed in dye tests are: (i) Indigo-carmine, (ii) phenolsulphonephthalein. These methods of testing renal function appear to find greatest favour with our surgical colleagues in assessing the operative risk in surgical conditions. They are obviously of use in determining which of the two kidneys is affected by disease after ureteral catheterization. There does not, however, appear to be exact information as to what function of the kidney is measured by the elimination of the dye, nor does there seem to be definite evidence that the results necessarily run parallel either with nitrogenous excretion or water elimination. Of the practical value of these investigations from the surgical standpoint there can be no question. The literature does not seem to indicate clearly the exact inferences to be drawn from dye excretion, and there is sometimes difficulty in correlating these findings with other clinical tests. The urologists present will no doubt be better able to shed light in these dark places.

In conclusion, it may therefore be stated that the tests of renal function, while of great clinical value and importance, cannot be used arbitrarily to define the actual state of renal damage. The tests are of relative value, indicating the degree of departure of kidney function from normal in various directions, and they should be interpreted with reference to the whole clinical picture presented by the patient.

References.

(1) T. Addis and C. K. Watanabe: "The Rate of Urea Excretion. II: The Rate of Excretion of Administered Urea in Young Healthy Adults on a Constant Diet," *Journal of Biological Chemistry*, Volume XXVII, 1916, page 249.

(2) H. Maclean: "Modern Methods in Diagnosis and Treatment of Renal Disease," Second Edition, 1924.

(3) E. H. Lashmet and L. H. Newburgh: *Journal of the American Medical Association*, Volume XCIV, Number 24, June 14, 1930.

(4) Volhard and Koranji: Quoted by T. C. Hunt, "Address on Recent Aids in Diagnosis of Some Common Disorders," *The Lancet*, October 12, 1929, page 751.

(5) H. O. Mosenthal: "Renal Function as Measured by the Elimination of Fluids, Salt and Nitrogen, and the Specific Gravity of the Urine," *Archives of Internal Medicine*, Volume XV, 1915, page 733.

RENAL EFFICIENCY TESTS AND THEIR SURGICAL APPLICATION.[1]

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THE question of the state of the renal efficiency is of major significance in the surgical patient and renal insufficiency of surgical importance can occur in several ways. A functional impairment

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 31, 1930.

of both kidneys may be present, due to a surgical lesion of the urinary tract or complicating a surgical lesion either here or elsewhere; or the impairment may be on one side only, due to local disease. Hence the estimation of renal efficiency, according to the requirements of the case, may be an estimation of: (i) the combined or total renal function; (ii) the separate or differential renal function.

ESTIMATION OF THE TOTAL RENAL FUNCTION.

Tests carried out for the purpose of estimating the total renal function are legion and fall into three groups: tests of retention, tests of excretion and correlation tests.

Tests of Retention.

Tests of retention consist of estimations, mostly quantitative, of the amounts present in the blood stream of various products of metabolism which are efficiently excreted by the healthy kidney, but retained to varying extents in disease. Such substances are fairly numerous, but present consideration will be confined to a few techniques that best combine simplicity, value and reliability.

Estimation of the Blood Urea.

Urea is a comparatively harmless end-product of nitrogenous metabolism which is excreted entirely by the kidney and is present in uniform concentration throughout the rest of the body. Under all conditions a certain amount of urea is present in the blood and in health this amount varies from 20 to 40 milligrammes per 100 cubic centimetres, though in the later decades of life values up to 50 milligrammes may be normal. When the kidney as a result of disease becomes unable to excrete the amount of urea produced daily, the blood urea commences to rise and its estimation therefore affords some index of the renal efficiency.

A high blood urea, however, is not an infallible sign of renal disease. In conditions in which the urinary output is low, such as cardiac failure or intestinal obstruction, it is of no significance unless accompanied by other very definite evidence of renal insufficiency.

Two factors may be mentioned which may detract from the value of the blood urea estimation. The first is that urea retention is not the cause of uræmia and a high blood urea therefore is not in itself synonymous with uræmia. On the other hand, urea excretion is one of the normal functions of the kidney and any information that we can get regarding the efficiency or otherwise of the performance of this function, must be of service. The second factor is the fact that the test is not a delicate one—in several types of renal disease it is only when anatomical changes have progressed to the point where the renal reserve power is practically used up, that the blood urea commences to rise.

This is well exemplified by Case I.

F., aged twenty years, was first seen two years ago, complaining of nocturnal enuresis, with some scalding on micturition, for four months. The urine was free from pus, X ray examination revealed no abnormality and cystoscopy showed a trabeculated bladder, with several cellules and gaping ureteral orifices. A cystogram was taken (see Figure I). The sodium iodide flowed up the ureters and gave a perfect picture of the renal pelves and ureters, in which a condition of very well marked hydronephrosis and hydroureter was present. Her blood urea at this time was 45 milligrammes per 100 cubic centimetres, showing what gross anatomical changes and how extensive an impairment of the functional reserve can be consistent with a comparatively low figure. The precarious condition of her renal function can be gauged from the fact that her blood urea is now 65 milligrammes, and the result of her urea concentration test is most unsatisfactory, as seen by the figures in Table I.

TABLE I.

Observation.	Maximum.	Minimum.
Range of urea concentration	1·2%	0·85%
Volume of hourly specimens	105 cubic centimetres	95 cubic centimetres
Specific gravity	1011	1009

Blood Creatinine.

Creatinine readings in excess of the normal figure of two milligrammes per 100 cubic centimetres occur from time to time in surgical renal disease, but since the kidneys can concentrate creatinine to a greater extent than is the case with urea, creatinine and urea retention do not go hand in hand and creatinine retention, when at all pronounced, is usually associated with well marked renal insufficiency.

Andrewes's Reaction.

Andrewes's test is a qualitative test of nitrogenous retention. It is a colour reaction given by uræmic sera and it is an absolute indication of very gross disease. It has been studied recently at the Royal Prince Alfred Hospital[1] in the laboratory animal and full confirmation has been afforded of the findings obtained clinically by workers elsewhere, that it is a very valuable test indeed in the diagnosis and prognosis of gross renal insufficiency. It is due to the retention of indican, of which the quantitative estimation also provides valuable information.

Tests of Excretion.

Tests of excretion can be subdivided into three groups: (i) Specific gravity and water tests, (ii) tests of urea excretion, (iii) tests of excretion of foreign substances.

Specific Gravity and Water Tests.

The simplest test of all is to allow the patient no fluids before he retires for the night and to estimate the specific gravity of the urine he passes on the following morning. This affords a rough measure of the extent to which he can concentrate the various products of urinary secretion; and, in general, the higher the specific gravity, the better is the renal function.

A second test, equally simple, is Volhard's concentration-dilution test.[2] The patient is given 1,500 cubic centimetres (two and a half pints) of water

in the early morning. He is expected to consume this in about three-quarters of an hour. The specific gravity of the urine should fall to about 1002 and he should excrete the water in about four hours. Later in the day the specific gravity mounts and should reach a value of 1025 late in the afternoon. The higher the difference between the specific gravities of the concentrated and the dilute specimens of urine, the better is the renal function, while, if the two figures approximate, the function is becoming dangerously impaired.

Tests of Urea Excretion.

Simple estimation of the resting urinary urea in the absence of a raised blood urea is usually of little assistance, as the findings provide no indication of the maximum concentration of urea that the kidneys are capable of excreting. Some form of urea concentration test is therefore generally made use of, the technique described by Maclean being usually followed.

The standard for the test, as laid down by Maclean,[3] is that if the concentration of urea exceeds 2% in the first two hours, the kidneys are doing their work efficiently, but this is not true in every case, for the following reasons:

1. Sometimes the urea causes so much polyuria that in the presence of a good renal function the concentration may not reach this figure.

2. It happens more often than not that the maximum concentration occurs in the third hour; for this reason a third hour specimen should always be taken.

3. By many 2% is regarded as too low a figure to be consistent with absolute normality.

4. In the interpretation of the test such factors as the extent of polyuria and the specific gravity are also important.

5. Since the concentration of urea varies with the blood urea to a certain extent, the estimation of this is essential in most cases. This point was brought out in the experimental nephritis recently produced in dogs at the Royal Prince Alfred Hospital[4]; practically every dog that died from uræmia, had *post mortem* urine in the bladder containing urea in excess of 2%. The highest value found was 4·63%.

Properly interpreted, the urea concentration test furnishes a useful test of renal efficiency. As with other tests, reliance should not be placed on it alone and it is but one of several factors which influence the final estimate of the patient's condition. Furthermore, it is not an accurate quantitative test, but rather a qualitative one.

Calvert has devised what he considers an improvement in the technique, with the object of estimating the range, rather than the actual value, of the urea concentration. I have collected a series of surgical patients at the Royal Prince Alfred Hospital who, by the courtesy of Dr. H. M. Rennie, have had urea concentration tests carried out, both techniques being used in succession. The series is small, but the Calvert technique appeared to provide no additional information and the results in some cases were anomalous and very difficult to interpret. The performance of the test upsets the ward routine rather more than does the Maclean technique and in the surgical patient it is, to say the least, not superior to the latter.

Tests of Excretion of Foreign Substances.

For the purpose of such tests by excretion of foreign substances, as a rule a certain quantity of the substance used is accurately measured and injected intravenously, sometimes intramuscularly, and the amounts excreted in the urine during each of the following two hours are estimated and expressed as a percentage of the quantity injected. These tests are therefore accurate quantitative estimations and for them to be reliable the technique must be above reproach. Injection must be purely intravenous and great care must be taken that the whole of the urine secreted in the two hours following injection is collected. If the presence of residual urine is at all possible, an indwelling catheter should be used. A good draught of water should be given just before the test; this causes some diuresis which makes the loss of small amounts of urine by errors in technique of less importance, while apparently not affecting the percentage excretion to an appreciable degree.

The objection to all these tests is that the excretion of the substances used is not one of the normal functions of the kidney, but the fact remains that several of them are very reliable, despite this academic objection, and will continue to be used.

Two groups of substances are used (i) dyestuffs, (ii) other chemicals. Of the latter, sodium thiosulphate is one of the most useful and reliable. A solution containing one gramme of the anhydrous salt is injected intravenously and 25% of this should be excreted within two hours, most of it in the first hour.

Dyestuffs: Phenolsulphonephthalein.—The phenolsulphonephthalein test was introduced by Rowntree and Geraghty[5] in 1910 and since then has come into general use. One cubic centimetre of 0·6% solution is injected and the percentage of dye excreted is easily and simply obtained, after alkalinization and appropriate dilution, either by comparison of the unknown with a series of standards or by the use of a colorimeter. It is stated that in health 60% to 80% should be excreted within two hours. These figures, however, are possibly somewhat high, since Walker-Taylor,[6] in a series of patients at the Royal Prince Alfred Hospital, all of whom were apparently normal, found that only about 20% reached 70%, while the average for the series was 63%. I think all figures above 50% should be considered as normal. The first hour's specimen should contain much more dye than the second. In fact, a detailed study of the test[7] has shown that in health the peak of elimination is reached in the first quarter of an hour, during which up to 40% may be excreted, falling rapidly to less

than 1% in the final quarter of an hour. In the presence of renal disease the percentage excretion, besides being diminished, is also delayed, a greater part of the dye being excreted in the second hour, so that the character of the curve of excretion departs from the normal. A study of this, however, would be too time consuming as a routine measure. It follows that since the excretion is delayed in the presence of renal insufficiency, the appearance time is also delayed and practically this is an important point. Equivalent information, however, is gained from a study of the percentage excretion and the appearance time with this dye finds its greatest use in the estimation of differential renal function, to be discussed below. This test is widely used and its value is established. It is worth while to mention that it is used as a routine, not only in surgical work, but in many medical clinics mostly in America. It is on the whole very reliable, though occasional anomalous results are reported, as, for instance, a low phenolsulphonephthalein excretion in the presence of normal responses to other tests, in such persons as prostatics *et cetera*. However, I should prefer to regard at all events some of these individuals as possessing some renal dysfunction not revealed by other tests. It is important to stress the fact that too much reliance should not be placed on a single unsatisfactory test; all such tests should be repeated with careful attention to technique.

To summarize, the objections to the test are: (i) it is easily influenced by errors in technique; (ii) it is not 100% accurate, errors of up to 10% quite possibly occurring in the reading. But the facts in its favour which outweigh these objections, are: (i) a 10% error is of little moment in the interpretation; (ii) its simplicity and wide applicability. The findings have to be correlated with those obtained by other means, as with all tests, but the phenolsulphonephthalein test, properly carried out, has the distinction in my opinion of being the most valuable single test in common use at present.

Indigo-carmine.—The indigo-carmine test differs from the phenolsulphonephthalein test in that the principal criterion for its proper interpretation is the appearance time, since there are serious technical difficulties in the way of quantitative estimation of the percentage excretion which furthermore is small, even in health, as a fairly large proportion of the dye is decolourized in its passage through the kidney. However, a fair idea of the rate of excretion can be obtained by simple inspection of the urine passed. In view of the importance of the appearance time, an indwelling catheter has to be used or cystoscopy carried out for accurate interpretation, so that the test has a somewhat limited application. In its own field, however, it is very valuable, so much so that in prostatic surgery it is possible to use it exclusively in the large majority of cases and not be led astray. Five cubic centimetres of a 0·4% solution are injected intravenously or a larger amount intramuscularly. The actual appearance time is four to five minutes. Specimens of urine are collected each fifteen minutes after injection.[8] The first specimen should be moderately coloured, the next richly so. With renal insufficiency, the appearance time is delayed up to an hour or more and the colour, when it does appear, is correspondingly poor.

Correlation Tests.

A third group of tests may be referred to by the name correlation tests. Urea is the basis of them all, the essential feature being an attempt to correlate the urea concentration of the urine with the blood urea. Various formulæ have been employed for this purpose, such as Ambard's coefficient and Maclean's index, which abound in square roots and decimals, but these are not used now to any great extent. A promising contribution to the problem has been made by Addis[9] who has devised what he terms the urea ratio test. By his technique he attempts to stimulate the whole of the functioning renal tissue into activity and by comparing the blood urea with the amount excreted in the urine in a given time, he claims that the resultant fraction is directly proportional to the amount of secreting tissue in the kidneys. His reasoning appears very sound, but the test is rather cumbersome for routine use.

The test which best combines simplicity and value is the urea concentration factor; it is simply the fraction

$$\frac{\text{milligrammes urea per 100 c.cms. urine}}{\text{milligrammes urea per 100 c.cms. blood}}$$

at any given time. This represents the number of times the kidney is concentrating urea at the time of examination and since the figures are available if the patient be having blood urea and urea concentration tests, they should be made use of. In health about seventy is the normal figure.

Application of Renal Efficiency Tests to the Surgical Patient.

Four types of renal insufficiency have to be considered:

1. That occurring in the surgical patient in general.
2. That due to bilateral surgical supravesical lesions.
3. That due to obstructions of the lower urinary tract.
4. Chronic nephritis complicating: (i) either of the two last mentioned classes of case, (ii) a surgical lesion of one kidney. This last section will be discussed as it arises in the consideration of (i) and (ii).

Renal Insufficiency in the Surgical Patient in General.

Some examination for the detection of renal insufficiency in the surgical patient in general should invariably be carried out. The simplest procedure is to examine the urine for albumin and casts and to estimate its specific gravity. If the results of all three tests are satisfactory, nothing more is

ILLUSTRATIONS TO THE ARTICLE BY DR. M. S. S. EARLAM.

FIGURE I.

Case I, bilateral pyelogram showing marked hydronephrosis and hydroureter. The blood urea in this instance was 45 milligrammes *per centum*.

FIGURE II.

Case V, right pyelogram. Estimation of differential renal function gave a very poor result. Nephrectomy was performed.

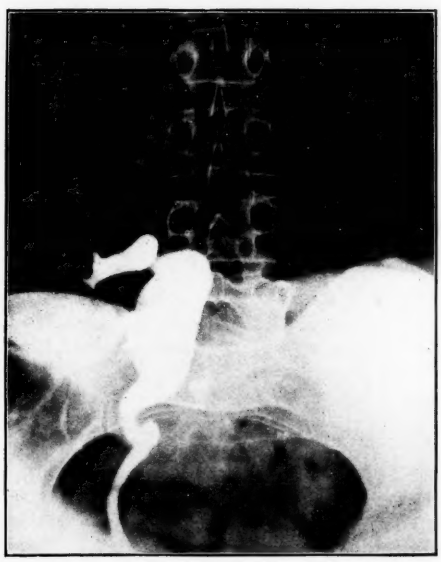

FIGURE III.

Case VI, right pyelogram. Compare with Figure IV showing the opposite side.

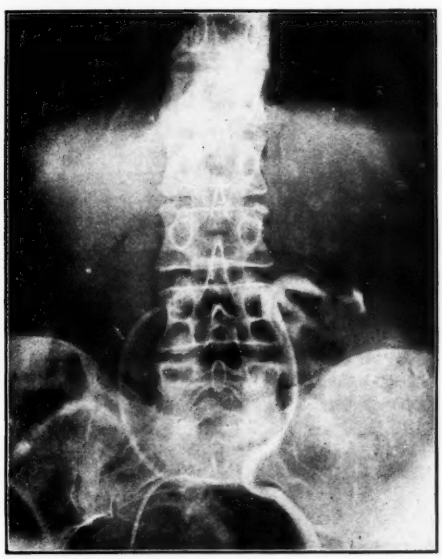

FIGURE IV.

Case VI, left pyelogram. The pelvis is incompletely filled, but the pyelogram demonstrates that this kidney is definitely smaller than its fellow, with almost certainly less secreting tissue.

usually necessary, but if any suspicion of renal disease arises, further investigation must be carried out to determine its extent. If there is any possibility that the renal insufficiency may have a surgical basis, for instance, if pyuria is present or the patient is a potential prostatic, this point must be decided by appropriate investigation. It happens usually that the contemplated surgical procedure can be proceeded with, but occasionally the insufficiency proves to be gross, when major surgical procedures, unless of an immediate life-saving nature, have usually to be abandoned.

Bilateral Supravesical Lesions.

The commonest of bilateral supravesical lesions is stone formation. In this connexion it may be mentioned that the stones which cause most damage, are not the large ones which may form casts of the pelvis and calyces, but the smaller ones which cause intermittent obstruction and hydronephrosis. The renal insufficiency that ensues is shown by raised blood urea and impaired urea concentration and excretion of dyestuffs; and the functional findings are in every way comparable with those of chronic interstitial nephritis. For instance, in a small series of ten case records which I was able to find in the Royal Prince Alfred Hospital records, the urea concentration test appeared to throw more light on the condition of the kidneys and to be more sensitive in the detection of the onset of renal insufficiency than did the blood urea. Out of the ten patients only one was able to concentrate urea to a percentage higher than two. This patient had a blood urea of 73 milligrammes per 100 cubic centimetres, had both sides operated upon and did well; while a second patient, with a much lower blood urea (49), but with a maximum urea concentration of 1·45%, was too ill for surgical interference.

Every patient with bilateral stone should have the renal function fully investigated and the functional findings should be correlated with the amount of anatomical change by pyelography. If the function is good or not very much impaired, bilateral operation may be safely carried out, but if the impairment is at all pronounced, it is usually contraindicated, as uræmia is liable to ensue after the anæsthetic and operative manipulations. Hard and fast rules cannot be laid down, as the clinical condition of the patient has to be taken into consideration and is a factor which is of equal importance with the functional findings. If infection be present, the function will be further impaired and such patients should be treated by conservative means (free fluids, pelvic lavage) in the hope that the function will improve sufficiently for operation to be done. With large staghorn calculi, operation is usually contraindicated, since they are not obstructive, cause little disturbance and the trauma involved by their removal is considerable.

Bilateral hydronephrosis due to causes other than stone is occasionally seen and treatment is necessarily palliative. Provided that the function is not hopelessly impaired, further progress of the condition can be frequently arrested by ureteral dilatation.

Bilateral polycystic disease of the kidneys gives a clinical picture identical with that of chronic interstitial nephritis. Surgical operation is usually not indicated, though improvement of function after Rovsing's operation (puncture of as many of the cysts as possible) has occasionally been reported.

Obstructions of the Lower Urinary Tract.

Chief of the obstructions of the lower urinary tract is hypertrophied prostate, with which is commonly associated some impairment, greater or less, of renal function. This has the unique feature of existing for considerable periods of time with no anatomical basis in the kidneys which are microscopically normal. Should the obstruction persist, anatomical change in the shape of hydronephrosis or infection may at any time occur and further impair the already disordered function. Furthermore, the fact should not be lost sight of that a certain proportion of patients will be found to be suffering from coexistent chronic nephritis.

The point of primary importance in the treatment of these patients is not so much the actual fact that the renal function is impaired, but the fact that the cause of this impairment, namely, the obstructive lesion, has forced the kidney to carry out its work under abnormal conditions. In other words, the renal function, besides being impaired, is also unstable, or rather has become unnaturally stabilized and must be reaccustomed to operating under normal conditions. Should prostatectomy be carried out as a primary procedure, the strain thrown upon the kidneys by the operation, the anæsthetic and the sudden alteration of their environment, may be sufficient to overwhelm them and cause the patient to die from uræmia. If, however, treatment be so directed that these necessary procedures be carried out at different times, the kidneys will stand an infinitely greater chance of proving equal to their task. In other words, the safest procedure is to reaccustom the kidneys to functioning under normal conditions by means of an indwelling catheter or a suprapubic cystostomy and remove the obstruction after a maximum of stabilization and efficiency has been obtained.

When the obstruction is relieved, the sudden alteration to normal conditions has the effect of further impairing the unstable renal function. Where the renal dysfunction has no anatomical basis; that is, if the kidneys are still anatomically perfect, this initial drop is succeeded by steady improvement till the results of functional tests, such as blood urea and dye excretion estimations, are normal in every way. If the function is little impaired, complete stabilization is usually attained in about ten days, but with grosser impairment a longer period of preparation is necessary. The extent of recovery is often remarkable, as shown by the following case, Case II.

M., aged sixty-eight years, had on admission to hospital a blood urea content of 167 milligrammes per hundred cubic centimetres. After twenty days' preparation by indwelling catheter the figure had fallen to 39 milligrammes per hundred cubic centimetres. At this stage radical operation was carried out and the patient did well.

Should much anatomical change be present, either as a result of long standing obstruction or due to coexistent chronic nephritis, the function will not return completely to normal, but since it is more important to obtain a stabilized than an absolutely normal function, operation can usually be done safely when this has been obtained. This is demonstrated by Case III.

M., aged sixty-nine years, on admission had a blood urea of 84 milligrammes per hundred cubic centimetres and a maximum urea concentration of 1·55%. After treatment by indwelling catheter for five weeks, his blood urea was still 71 milligrammes per hundred cubic centimetres and his urea concentration worse than ever, his maximum concentration being less than 0·5%. In other words, his renal function was neither satisfactory nor stabilized. At this stage a cystostomy was carried out and he was sent home for three months. At the end of this time his blood urea was 69 milligrammes per hundred cubic centimetres and maximum urea concentration 1·27% and three months later they were 68 milligrammes per hundred cubic centimetres and 1·45%. His function, though far from satisfactory, was stabilized, prostatectomy was carried out and the patient did well.

It occasionally happens that a grossly impaired renal function, after insertion of an indwelling catheter, will, after the initial drop, keep on falling instead of improving, so that the patient dies from uræmia. This was seen in Case IV.

M., was seventy years of age. His blood urea on admission to hospital was 113 milligrammes per hundred cubic centimetres. After eight days of catheter drainage it was 344 milligrammes per hundred cubic centimetres, and seven days later he died from uræmia.

After this brief account of the nature of the renal insufficiency that accompanies the hypertrophied prostate, and the various results that may follow the sudden relief of the obstruction, it becomes necessary to discuss the routine treatment of the renal insufficiency, that is, the application of the various renal efficiency tests.

The first question to be decided is: Is it safe to insert the indwelling catheter and is the possibility of subsequent uræmia, as in the last case quoted, at all likely? It is impossible to lay down definite rules, but several factors may be of help. (i) Careful consideration should be given to the patient's general condition. If he has a good colour and looks fit, it is unlikely that much dysfunction exists and the insertion of the catheter will probably be safe. (ii) If the routine physical examination reveals a distended bladder, it should on no account be emptied at one sitting, as uræmia is very liable to supervene. A catheter should be tied in and the bladder be emptied gradually over a period of days or some form of gradual decompression apparatus be used. (iii) It seems to me very likely that one of the factors in producing a sudden uræmia after insertion of the catheter is infection, to which these patients, with their lowered general resistance, are very prone. The very strictest attention should be paid therefore to technique. (iv) Are the renal efficiency tests of any assistance? It is commonly stated that there is no way of differentiating renal insufficiency due to hypertrophied prostate from that due to chronic nephritis. One important point, however, is that polyuria is not a feature of the former condition. In both conditions the urea concentration factor falls, but whereas with chronic nephritis the kidney compensates for this by passing large quantities of urine with a low urea content, with hypertrophied prostate there is in general a tendency for the blood urea to rise, while the urinary urea is normal and the result of the urea concentration test good. I have confirmed this point in a series of two hundred cases in the Royal Prince Alfred Hospital. So that if the urine has a good urea content or high specific gravity, it is rather an indication of the absence of anatomical change and the patient's outlook on the whole is good. A further point is that very frequently the blood urea is found to be raised out of all proportion to the creatinine. If the creatinine content is very much raised, the patient must be treated with great caution. If suspicion arise at all as a result of any of the above considerations, the only tests it is safe to employ are the retention tests, that is, blood urea, blood creatinine and Andrewes's test, and simple examination of the urine for urea content, casts and specific gravity. If the onset of uræmia appears a definite possibility, the safest procedure is to insert the catheter and decompress the kidneys gradually.

Having proceeded thus far, the next point to be decided is: When is the patient ready for radical operation? To decide this, the functional tests, of whatever nature, must be carried out regularly, as it is far less important to know the patient's actual function at any one time than the extent of his progress and the degree of stabilization. In settling this question, the estimation of the blood urea and dye excretion are the most valuable tests, the former gradually falling and the latter rising; but, on the other hand, the urea concentration test, while useful in the first evaluation of the patient's function, provides little information as to his progress in the majority of cases, either remaining stationary throughout the period of preparation or actually dropping as the blood urea falls and the function improves. The test being qualitative rather than quantitative, accurate deductions as to improvement cannot be made from it. Operation should be deferred till a maximum of efficiency and stabilization are present, with a good twenty-four hours' excretion, whether this takes a week or many months. As shown previously, it is impossible to lay down rules regarding safety of operation in terms of blood urea or phenolsulphonephthalein, as a stabilized, rather than a completely normal, function is the desideratum. In such cases it is important to remember that a single test, unless it be a dye test, should not be relied on. No two tests provide exactly similar information and they vary in sensitivity. More than one test should be used and within reason the more the better.

Lastly, it should be emphasized that renal insufficiency is only one of the dangers of hypertrophied prostate and the results of the efficiency tests are only a part of the composite picture, into which the patient's age, physical condition, respiratory and cardio-vascular conditions and the presence or absence of secondary infection, all enter; but if by the means described above the renal function can be brought to a stage where it will permit of radical operation with the danger of uræmia reduced to a minimum, it is a big step forward towards the ultimate cure.

In concluding this section, it should again be emphasized that a combination of tests, rather than a single one, should be relied on. In the case of the dye tests alone it is possible in most urological cases to carry out safe surgical operation without calling in other laboratory aids. These tests are very sensitive and stand above other methods in that a satisfactory excretion provides positive evidence of a good renal function, but they have the drawback that, as the amount of renal dysfunction gradually increases, the dye excretion drops rapidly to a minimal amount, and other methods have to be employed to demonstrate the extent of the renal insufficiency shown to exist. The best combination of tests at our present disposal is that of blood urea, urea concentration and either of the two dye tests, of which I prefer the phenolsulphonephthalein. All these furnish different information and each supplements the others. Other tests, as the blood creatinine, concentration-dilution test *et cetera,* should be employed from time to time as the indication for their use arises.

Estimation of Differential Renal Function.

Technique.

The technique of estimating the differential renal function is as follows.

1. Estimation of the urea content of the individual kidney urines obtained by ureteral catheterization. The patient is given fifteen grammes of urea two hours prior to cystoscopy. This test, though quite useful, has found less favour than the dye tests, principally because the patient can be allowed no fluids before cystoscopy, so that the collection of the specimens will usually be tedious and the dye tests are more simply performed.

2. Phenolsulphonephthalein test. The usual dose of one cubic centimetre of 0·6% solution of phenolsulphonephthalein is injected intravenously after catheterization of the ureters and the specimens of urine are collected into an alkaline solution. Two criteria are made use of in interpreting the test: (i) Appearance time. This should be four minutes or less and it is remarkable how little this varies in health. The size of the catheter used and the presence or absence of diuresis appear to have little influence. In the presence of disease the appearance time is always lengthened. (ii) Percentage excretion. Comparison between the amounts of dye excreted by the two kidneys can be made over periods of a quarter or half an hour. An good deal of error can arise in this estimation owing to leakage around the catheters and inequality of rate of flow through the catheters, but provided that both catheters are of equal size and fairly large and have both entered the pelvis and are running well, satisfactory conclusions can be drawn.

3. Indigo-carmine test. As previously mentioned, the appearance time is the main criterion with the indigo-carmine test and it has the advantage that the ureters need not be catheterized, the blue efflux from the orifices being clearly seen. On the other hand, if the excretion is delayed, it is more unpleasant for the patient to lie still with the cystoscope *in situ* while the surgeon waits for the dye to appear, than is the case when ureteral catheters have been passed and the cystoscope removed. Of course the indigo-carmine test can be used just as well as the phenolsulphonephthalein after ureteral catheterization. I have been accustomed to use the latter test and have made use of the indigo-carmine test only in the occasional case where the ureter cannot be catheterized owing to angulation in its intramural part. In such cases it is the only means we have of estimating the differential renal function.

Between the phenolsulphonephthalein and the indigo-carmine tests there is little to choose. Both tests, properly applied and interpreted, are very reliable. One important point in their application is that the patient should not be under a general anæsthetic, as the excretion is often definitely inhibited thereby, nor under a spinal, for the same reason.

Application of the Above Tests.

The estimation of differential renal function is carried out to answer one of three questions, namely:

1. Which kidney is diseased? It happens from time to time that this question can be answered, apart from double pyelography, only by this procedure and the commonest condition requiring it is that of painless hæmaturia, seen in an interval between attacks, for which no cause in the lower urinary tract can be found. A delay in the appearance time of the dye on one side will often point the way to the seat of the lesion, the nature of which will be disclosed on further investigation.

2. Has the function of this kidney become so impaired that it requires removal? This question is important because the work of Hinman[10] on renal counterbalance has shown that after a certain amount of renal damage has been sustained, recovery is not possible. The explanation for this is that if compensatory hypertrophy has occurred on the opposite side and the function taken over, the damaged kidney has no stimulus to recover and a conservative operation is useless. Practically, however, this question is usually better answered by X ray, pyelography and microscopical and cultural examination of the kidney urine than by functional tests. It happens in some cases that these findings

are not conclusive, when a differential function is of much help. This occurred in Case V.

F., aged forty-six years, complained of pain in right loin and urinary frequency and scalding for two years. Physical examination revealed pyuria, but nil else abnormal. X ray examination revealed no abnormality. The right kidney urine contained pus and yielded a culture of *Staphylococcus albus*, the left kidney urine was pus free and sterile. The right pyelogram (Figure II) showed a stricture at the uretero-pelvic junction, with some clubbing of the calyces. The appearance was not entirely conclusive. A differential estimation of renal function was carried out, phenolsulphonephthalein being used, with the following findings. On the left side the appearance time was two minutes, 16% secretion in fifteen minutes. From the right kidney the appearance time was six minutes, 2% secretion in fifteen minutes. On these findings a right nephrectomy was carried out. Microscopically the kidney was grossly disorganized, with extensive fibrosis and small round cell infiltration.

3. Will this kidney support life efficiently if its fellow be removed? It is possible in many cases to hazard a guess that the answer is in the affirmative, if one side is completely disorganized and the combined renal function is normal. One has to be very sure, however, that disorganization is complete, since a good deal of renal tissue can be destroyed with little alteration in the functional findings, and it is quite possible that removal of a badly damaged kidney which even so contains most or all of the actively functioning renal tissue, may unknowingly be contemplated. Such assurance can be given only by a very complete investigation, in the course of which the good function of the opposite side can be confirmed with very little extra trouble.

If the operation of nephrectomy is contemplated, or is even a remote possibility, the function of the opposite side should always be investigated, for the following reasons:

1. The patient may have a congenital solitary kidney. This is not common, but crops up every now and again. Figures collected from autopsy statistics[11] record the frequency as one in 2,400, but the probability of encountering the condition is intensified by the findings of Anders,[12] who in a collected series of 170 cases found pathological changes in 79. I have seen five cases in the past four years, in all of which the diagnosis was established beyond doubt at operation or autopsy. Suspicion should arise if no ureteral orifice can be found on the presumed good side or if total obstruction to ureteral catheter or iodide injected be present a short distance from the bladder. The complete absence of any excretion of indigo-carmine will make the diagnosis very probable and at any rate will completely rule out radical operation. The absence of a kidney shadow in a good X ray film is also of help.

2. The patient may have an atrophic kidney, due either to disease or to congenital hypoplasia. This condition is important, since such kidneys are usually unable to sustain life after opposite nephrectomy. It is quite easy to miss the diagnosis, since the kidney may be histologically normal, resulting in a urine of normal composition and unchanged appearance time of phenolsulphonephthalein and indigo-carmine. It can be diagnosed by the fact that the quantitative excretion of phenolsulphonephthalein is markedly impaired. This is highly suggestive, seeing that such a finding due to disease is practically always accompanied by a delayed appearance time. A pyelogram will establish the diagnosis. This condition was seen in Case VI:

F., aged sixty-three years, gave a history of right lumbar pain with urinary frequency and scalding for six years. Her right kidney was infected with *Bacillus coli communis* and a pyelogram (Figure III) showed a large extrarenal pelvis and a very unusual appearance suggesting a double ectopic kidney. Her left pyelogram (Figure IV) showed a much smaller kidney and her left kidney urine was of normal composition. She had the greater part of her functioning renal tissue on the right side, which was the diseased one. Fortunately she was suffering from a simple pyelitis, which responded well to the usual measures.

3. The kidney may be affected by disease. In such an event the appearance time of the phenolsulphonephthalein or indigo-carmine will be prolonged and the concentration poor. If the former dye be used, quantitative estimation is not necessary, as the poor concentration is usually obvious. If these findings are present, three posibilities exist.

The first possibility is that the disease for which the nephrectomy was planned, may be bilateral. This is most frequently the case in infective lesions. In such an event the diagnosis will be established by cultural and microscopical examination of the kidney urine and pyelography. If the lesion is at all well marked, conservative measures must be adopted, but it occasionally happens that the infection may be very early on one side and very gross on the other, when the early lesion will usually clear up after the bulk of the trouble has been surgically removed. Even with renal tuberculosis, occasional patients are seen with a gross lesion on one side, with toxic symptoms and an early lesion on the other, which improve tremendously after nephrectomy and appear to overcome the remainder of the infection.[13] If, however, any antomical changes are evident in the pyelogram, radical surgical operation is contraindicated.

The second possibility is that a condition of toxic nephritis may be present. This will be the case only if the opposite kidney is the seat of a gross infective lesion, such as a pyonephrosis or renal tuberculosis, either with or without secondary infection. Such patients look sick and are anæmic, but the pyelogram of the good side is normal and the urine from that side pus free and sterile. Tests of total renal function showed definite impairment. This condition cannot be distinguished with certainty from chronic nephritis, but the toxic appearance of the patient is always suggestive; and seeing that the diseased kidney is usually quite disorganized, the question of the presence or absence of chronic nephritis does not affect the treatment. These patients improve wonderfully after nephrectomy, the function gradually returning to normal. Such a condition was evident in Case VII.

F., aged thirty-eight years, gave a history of pain in left loin for five years. On examination there was a marked

swelling in the left lumbar region and gross pyuria. Temperature fluctuated between 36·7° and 39·4° C. (98° and 103° F.) and the patient looked and was very ill.

The left pyelogram showed complete disorganization, there being a condition of tuberculous pyonephrosis. The right kidney urine was pus free and sterile on culture. The blood urea was 136 milligrammes per hundred cubic centimetres, creatinine 1·95 milligrammes per hundred cubic centimetres, the maximum urea concentration 1·95%, while phenolsulphonephthalein appeared in five minutes from the right kidney in fair concentration only. Nephrectomy was carried out and the patient never looked back.

The third possibility is that the patient may be suffering from chronic nephritis. Such a condition should be suspected if the results of the function tests are poor without the presence of either the toxic element just described or demonstrable anatomical change. Tests of total renal function should be made, the urine examined for albumin and casts and the blood pressure taken. The presence of chronic nephritis having been established, the case has to be reviewed on its merits. If there is much functional tissue in the kidney it was proposed to remove, it will probably be better to carry on with palliative treatment or perform a conservative operation, but if the kidney is completely disorganized or the seat of a new growth, it is better removed. As with other conditions dealt with in this paper, no hard and fast rules can be laid down. Case VIII illustrates the occurrence of this complication.

M., aged twenty-five, complained of intermittent left lumbar pain for nine months. He had no urinary symptoms. Physical examination revealed no abnormality except for a few pus cells per high power field. Urine was sterile on culture and repeated examinations failed to reveal tubercle bacilli. Cystoscopy showed a normal right ureteral orifice, while the left one was so retracted that it could not be seen and catheterization was impossible. Exploratory operation on his left kidney was contemplated. His right kidney urine was free from pus and sterile on culture. Phenolsulphonephthalein appeared in five minutes from this side and the concentration was definitely poor. A right pyelogram was normal. His blood urea was 65 milligrammes per hundred cubic centimetres and resting urinary urea 0·8%, giving him a urea concentration factor of only 12·3. His symptoms not being very incapacitating and his occupation sedentary, it was decided that he was better off carrying on as he was.

I have mentioned the various conditions which may cause an impaired excretion of dye by the supposedly healthy kidney. The opposite finding is quickly disposed of. A normal appearance time, followed by a rich secretion of dye, provides positive assurance that the kidney concerned will support life efficiently if its fellow be removed. To this rule I have seen no exceptions.

It appears by the above remarks that by complete investigation and application of either of the two standard dye tests when indicated, a complete understanding of the state of the differential renal efficiency will be obtained and the findings will point the way to the line of treatment, conservative or radical, which is in the patient's best interests, and the possibility of disaster from renal insufficiency after nephrectomy be eliminated. Practically this is the case and I have seen no single instance in the past four years where the result of a dye test, correctly interpreted, proved unreliable in the estimation of differential renal function.

REFERENCES.

(1) Adolph Bolliger and M. S. S. Earlam: "The Andrewes Diazo Reaction: Its Presence and Significance in Experimental Renal Insufficiency," THE MEDICAL JOURNAL OF AUSTRALIA, April 12, 1930, page 474.

(2) F. Volhard: *"Ueber die funktionelle Unterscheidung der Schrumpfnieren," Verhandlungen des Deutsches Kongresses für innere Medizin, Band* 27, 1910, *Seite* 735; quoted by A. M. Fishberg, *Journal of the American Medical Association*, Volume LXXXVIII, June 25, 1927, page 2018.

(3) H. Maclean: "Modern Methods in the Diagnosis and Treatment of Renal Disease," 1921.

(4) Adolph Bolliger and M. S. S. Earlam: "Experimental Renal Disease Produced by X Rays: Production and Functional Study of a Standardized Lesion," THE MEDICAL JOURNAL OF AUSTRALIA, March 15, 1930, page 300.

(5) L. G. Rowntree and J. T. Geraghty: *Journal of Pharmacology and Experimental Therapeutics*, Volume I, 1910, page 575.

(6) P. N. Walker-Taylor: Personal communication.

(7) E. Clay Shaw: "A Study of the Curve of Elimination of Phenolsulphonephthalein by the Normal and Diseased Kidneys," *Journal of Urology*, June 13, 1925, page 575.

(8) Frank Kidd: "Tests of Renal Efficiency," *The Lancet*, April 22, 1922, page 788.

(9) T. Addis: "Renal Function and the Amount of Functioning Tissue," *Archives of Internal Medicine*, September 30, 1922, page 378.

(10) Frank Hinman: "Renal Counterbalance," *Archives of Surgery*, June, 1926, page 1105.

(11) E. Ballowitz: *"Über angeborenen einseitigen vollkomenen Nierenmangel," Virchow's Archiv, Band* CXLI, 1895, page 309.

(12) J. M. Anders: "Congenital Single Kidney with the Report of a Case: The Practical Significance of this Condition, with Statistics," *American Journal of the Medical Sciences*, Volume CXXXIX, 1910, page 313.

(13) A. W. Adams: "The Rôle of Pyelography in Tuberculous Nephritis," *British Journal of Urology*, March 2, 1930, page 36.

Reports of Cases.

OSTEOCHONDRITIS DISSECANS.

By S. H. SCOUGALL, M.B. (Sydney),

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A.B., AGED nineteen years, attended the out-patient department on September 22, 1928, with a history of knee trouble since 1922, when he ran a stick into the inner aspect of his right knee. About six months later a piece of wood was removed from the area. Periodically since, the knee has become swollen and painful and this has been accompanied by locking, with tenderness to the medial side of the patella. External rotation of the tibia on the femur, with flexion of the knee, was the most painful movement. He had at the time no limitation of flexion or extension of the knee.

A diagnosis of a torn medial meniscus was made, but X ray examination revealed two large cartilaginous loose bodies present. The condition appeared to Dr. H. R. Sear to be one of intracapsular chondromatosis; slight osteoarthritis was present.

At operation on November 12, 1929, some small white bodies were obtained through a vertical incision to the inner side of the patella, but no large body could be found. A postero-lateral incision into the joint also disclosed no further loose body, but a postero-medial approach showed a large flattened loose body, roughly circular, somewhat bigger than a shilling, one side cartilaginous and the other side resembling cancellous tissue.

This body was attached by a fibrous band to the postero-lateral aspect of the lateral epicondyle of the femur. It must have wandered across the anterior compartment before finally lodging in the postero-medial compartment. The lateral epicondyle in the area of attachment of the fibrous band showed an equivalent area which had the appearance as if this large flake had been chipped off. The wandering of the loose body would appear unique.

The second body shown by X rays could be felt through this excision to be extracapsular, and was not removed. The small loose bodies consisted of fibrous tissue without cartilage and the large body showed both bony and cartilaginous structure.

The patient has full mobility of the knee and has indulged since in strenuous sports.

It is becoming generally established that trauma plays a chief part in the ætiology of *osteochondritis dissecans*. Another point of interest is the rarity of the location of the bone flake from the postero-lateral aspect of the lateral epicondyle, the usual location being the lateral aspect of the medial epicondyle.

The condition generally occurs in males of between fifteen and twenty-five years of age, as would be expected, and as many as three loose bodies have been found in a joint. The knee is the joint commonly affected, but these bodies are said to occur in the hip, elbow and other joints as well.

CÆSAREAN SECTION: RUPTURE OF THE UTERUS WITH CONSERVATION OF THE UTERUS.

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Case I.

MRS. A.B., aged twenty years, was due to be confined on July 1, 1924. The head was not engaged at the onset of labour which started two days before the estimated date. Soon after labour started the pains became violent and the cord prolapsed and as the patient was being confined in her own home, a quick Cæsarean section was impossible. I failed to replace the cord and did a podalic version. The child was still-born.

Fourteen months later labour was due again. She was given quinine and castor oil treatment two weeks before term, but as the head failed to engage after five and a half hours' labour in which the pains became violent as in her first labour, a Cæsarean section was done and a male child, eight pounds, was delivered.

About two years later, when the patient was twenty-four days off term, I was informed she had some abdominal pains and constant desire to pass urine. When I saw her she was feeling very uncomfortable, but there were no signs of shock. On palpation I could feel the fœtus abnormally easily through the abdominal wall in the region of the previous scar. Judging that the uterine scar had ruptured while the membranes had remained intact, I asked that the abdomen should not be palpated in the hospital where I sent her for operation.

The abdomen was opened in the line of the previous scar, the whole length of the uterine scar had ruptured and the intact membranes presented through the wound. A living baby, 2·4 kilograms (five pounds six ounces), was delivered.

The condition of the patient was good. The old edges of the uterine wound were trimmed, the fibrous borders being removed and the new edges approximated by interrupted stitches. The patient was sterilized. She made an uninterrupted recovery.

Comment.

The uterus was thus retained, enabling the patient to menstruate as usual, an important psychological factor in a young woman of twenty-three years. Moreover, removal of the uterus, in spite of leaving the ovaries, tends to cause an early menopause.

Case II.

Mrs. C.D., aged twenty-six years, was a patient on whom I performed a Cæsarean section after a trial labour for her first pregnancy. One morning three years later I was called by her medical attendant who stated that she had suddenly collapsed, after being in labour for some time.

In spite of her history, she failed to seek antenatal care and did not call medical aid until she had been some time in strong labour.

A diagnosis of ruptured Cæsarean scar having been made, the abdomen was immediately opened; free blood filled the peritoneal cavity, but the Cæsarean scar was intact; this was incised and a dead child, 4·08 kilograms (nine pounds ten ounces) was delivered. A Y-shaped tear was discovered in the lower uterine segment. This and the incision in the upper segment were rapidly sutured, as her condition was desperate. The patient made a good recovery.

Comment.

The case is an example of the known fact that at times tonic contraction followed by rupture of the lower uterine segment will take place while the scar in the upper segment remains intact in a patient who has had a previous Cæsarean section. As in the previous case the uterus was saved.

Reviews.

A MANUAL ON NURSING.

THE fact that a third edition of "Theory and Practice of Nursing," by Miss M. A. Gullan, has appeared so quickly after the publication of the second is sufficient evidence that the instruction therein given to nurses has proved of value.[1]

In the preface Miss Gullan points out that the original text has been little altered, but that some additions have been made to include fresh data and emphasize details of nursing methods. The chapter on elementary dietetics has been amplified in accordance with recent knowledge and now follows the chapter on food and feeding.

It is a matter for regret that Miss Gullan still adheres to the somewhat "low" number of calories needed for the daily diet of the average working man as accepted in Australia, and that she has not seen it advisable to point out the necessity of a diet for rapidly growing adolescents even greater in amount than that required by many adults. Apart from these drawbacks, the chapter is excellent, while the coloured diagrams which accompany the chapters on digestion and absorption are of the greatest assistance in making diagrammatically clear the processes of assimilation of food.

Two new appendices have been added, one on food tests and digests, the other on common abbreviations in prescriptions—both most useful. An error has crept into the numbering of these: they are not consecutive, as is stated in the preface.

The interleaving of the book with blank pages for notes should enable nurses to add greatly to the value of the volume from their own experience. There is no doubt that much useful knowledge is forgotten and so lost, because not written down and passed on to others.

The admirable way in which Miss Gullan imparts the knowledge shown in every chapter of the book and more especially in those on the detailed methods of nursing, abundantly proves that she must have been a painstaking and accurate observer, with the gift of passing on her gains to others.

The book is one which can be strongly recommended to all nurses, whether probationers or sisters; even the most experienced will find it repay careful study. It should have a place in the library of every hospital and more especially in those which are training schools.

[1] "Theory and Practice of Nursing," by M. A. Gullan, S.R.N.; Third Edition; 1930. London: H. K. Lewis and Company, Limited. Demy 8vo, pp. 262, with illustrations. Price: 9s. net.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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RENAL EFFICIENCY TESTS.

PROBABLY no subject is arousing so much interest at the present time among all medical practitioners, physicians and surgeons, clinicians and laboratory workers, as that of renal efficiency tests. The physician wants to obtain definite information of the state of his patient's kidneys, so that he may order his patient's life in such a way that his comings and goings will put no strain on his excretory organs that cannot be easily withstood. The surgeon as a rule wishes to determine the ability of the patient's renal function to cope with the stress of a surgical procedure, particularly if the offending lesion involve the urinary tract. The biochemist is concerned with the chemical composition of the blood and of the urine in health and disease and with the study of the normal processes of excretion. His knowledge is of basal importance, he supplies the torch that will shed light in dark places. It is for this reason that the paper by Dr. F. S. Hansman is first of a series of three appearing in this issue. These three papers on renal efficiency tests are worthy of careful study. Dr. Holmes à Court puts the physician's point of view with an erudition commensurate with his vast clinical experience, and Dr. Earlam, well known to readers of this journal as an investigator in the field of urology, gives an excellent presentation of the surgical view.

Several points of importance emerge from the papers and from the discussion, and call for emphasis. In the first place the physiological process of renal excretion is not understood. As Dr. Hansman states, until the physiology, pathology and morbid clinical states of renal diseases can be brought into a common relationship, all tests for renal function will remain unsatisfactory. In the second place the fact that satisfactory results have attended the use of certain tests does not necessarily indicate that the kidney is normal; many factors have to be considered. Excretion may be carried out completely in spite of severe functional impairment of the kidney, by compensatory polyuria or because the intake is sufficiently low to be within the lessened capacity of the damaged kidney. It must also be borne in mind that deficient excretion of a substance in the urine is not necessarily the fault of the kidney itself; for example, diminished excretion of water and chloride in the urine may result from myocardial weakness, from the presence of ascites in cirrhosis of the liver, from vomiting *et cetera*. In other words, renal efficiency tests must be regarded only as an indication pointing in a certain direction; they must be considered in conjunction with the other clinical findings.

There is a danger in considering tests of function that undue prominence may be given to function, that attention may be diverted from the importance of structure. Dr. A. H. Tebbutt did well to raise this point in the discussion on the three papers. Function is so dependent on structure that the one should not be considered apart from the other. Dr. Tebbutt called attention to the recent work by Dr. Dorothy Russell, coming from the laboratory of Professor Turnbull, and published by the Medical Research Council. A special abstract of this investigation is published on another page in this issue. Numerous classifications of nephritis of a more or less satisfactory nature have been made. While Dr. Russell's classification need not necessarily be accepted in its entirety (it is perhaps somewhat unwieldy and Dr. Russell practically admits this), it certainly fits both the clinical and histological findings. It also shows that if further advance is

to be made in the elaboration of tests for renal efficiency, it must be made by correlating the clinical with the histological findings. Opportunities for carrying out histological examination in the early stages of renal impairment after complete investigation by laboratory methods are admittedly rare, but when they do arise, they should be eagerly followed; indeed, we can go further than this and add that the opportunity should be sought. One possible result of such correlation would be the throwing of light on the physiological process of renal excretion in the normal individual.

Since this journal is devoted primarily to the interests of the general practitioner, the subject of renal efficiency cannot be left without reference to the value of renal efficiency tests in prognosis. The prognostic value of these tests is greater in chronic than in acute renal disease and in so-called medical conditions than in those which are best treated by surgical measures; in the latter immediate relief often follows the performance of an operation. Such tests as the urea concentration test are not difficult to perform and they will be found of immense value in general practice.

Current Comment.

ACIDOSIS AND DEBILITY.

A STATE of constant rude health is one which few, if any, people are able to maintain. All at some time or another are affected to a greater or less degree by the absence of a sense of well-being. They complain that they are run down or feel physically and mentally below par. No organic lesion can be found nor is there evidence of infection, but the physiology of the organism has lost some of its efficiency and a state of subnormal health may be said to exist. Tonics and vacations are prescribed with great frequency and considerable success, but scientific investigation has been lacking. The dangers of the condition can be readily conceived; its significance is not appreciated. The term "lowered resistance" is often used in association with a condition of subnormal health, but it would appear that few have thought to ask why the resistance should be lowered or precisely what abnormality or abnormalities of function might be concerned. The inquiring mind should not be content with the generalizations of those who airily dispose of the problem by explaining that it is merely a matter of fatigue. It is remarkable that a condition which bulks so largely in everyday medical practice should have received so little attention from the investigator; perhaps its very familiarity is responsible.

In a recent report entitled "Acidosis and Debility: A Contribution to the Study of the State of the 'Soil' Disposing to Disease," A. Arnold Osman and Harold G. Close draw attention to the possibility of gaining valuable knowledge by means of a biochemical study of people in a subnormal state of health.[1] They define acidosis as a reduction of the sodium bicarbonate content of the blood plasma and use the extent of the reduction as a relative measure of the acidosis. The use of the term acidosis, of course, does not necessarily imply that the blood has become acid in reaction; indeed Osman and Close did not estimate the hydrogen ion content at all in this investigation. There was no evidence of ketosis.

They investigated normal adult males, normal, pregnant and debilitated females, and normal and debilitated children. The normal adult males were all healthy medical students, the normal adult females were trainees at a massage school, and the normal children were selected from those attending a hospital out-patients' department for such a local condition as ringworm, for example. The debilitated were also hospital out-patients. Debilitated men were not included in the experiment, as men usually attend hospital only on account of definite disease. The highest average content of sodium bicarbonate was observed in normal adult males, the next highest in normal adult females during the intermenstrual periods, the next in normal children, then in descending order in normal adult females during menstruation, in debilitated children, in debilitated adult females, and lowest in normal pregnant women. The quantity of water in the body appeared to vary inversely with the sodium bicarbonate content, that is, it increased with the acidosis. The question might then be asked to what degree dilution affected the percentage of sodium bicarbonate in the blood and whether there was an actual decrease in the total quantity of sodium bicarbonate in every instance in which an apparent decrease was noted. Widal remarked that œdema may be recognized clinically only when six kilograms of excess water are retained in the body; such a quantity might be capable of causing a considerable dilution of the blood. In pregnancy excessive fluid is retained, and it might be suggested that this to some extent may account for the low percentage of sodium bicarbonate in the blood of the parous woman.

The percentage of sodium chloride in the blood was found to vary inversely with the sodium bicarbonate percentage, save that it was high in normal children and highest in subnormal children. This fact only increases the complexity of the problem.

Finally, the authors conclude that many published figures concerning the acid-base balance in various diseases are of doubtful value, as frequently estima-

[1] *The Quarterly Journal of Medicine*, July, 1930.

tions have been carried out on patients of the "hospital class," the normal for whom has hitherto never been established.

Osman and Close deserve congratulation on their work; it gives a lead for others to follow in the exploration of an important and a readily available field for research. At the same time it is well not to lose sight of the fact that many debilitated people in whom clinical examination reveals no lesion, are actually suffering from some organic disease.

Surely progress towards the lofty ideal of disease prevention can never be great while it is hampered by a lack of knowledge of the soil on which disease is apt to flourish.

EMOTIONAL HYPERTENSION.

THE association of a temporarily raised arterial blood pressure with the nervous person's first visit to the physician is a circumstance with which every medical man is familiar. It merely serves to demonstrate the great variations possible in blood pressure in the absence of arterial disease. In many instances the pressure may actually be observed to fall while the sphygmomanometer cuff is still in position. The mechanism controlling the blood pressure in health and disease is imperfectly understood. The significance of abnormally high blood pressure has been the source of considerable controversy which has not provided a great deal of enlightenment.

In a recent paper Edward J. Stieglitz discusses what he terms "emotional hypertension."[1] He points out that the blood pressure may increase with emotional stimuli both in the sleeping and waking states, but that as a general rule such increase has no ill effects save in individuals whose arterial tension is already above normal. In some individuals, however, vasomotor control is so unstable that on the application of a suitable stimulus the blood pressure rises to such a degree as to produce subjective symptoms and to be a definite menace to health. These individuals may frequently be found to have some sexual abnormality, such as impotence or excessive *libido*, and in fact emotion of an erotic nature may be the only stimulus capable of inducing an attack of hyperpiesia. Disorders of the endocrine glands may have some ætiological bearing on the condition, but Stieglitz discovered no evidence of dysfunction of thyreoid or adrenals. There is no apparent organic disease of the heart and there is usually a normal response to effort. No doubt an increase in the pulse rate accompanies the increase in blood pressure, but he makes mention neither of this nor of the condition of the heart during the attack. He suggests the possibility that recurring attacks of emotional hypertension might eventually result in permanent hypertension and points out that the majority of persons suffering from high blood pressure have the so-called "nervous temperament." He states that the anatomical changes which occur in the arterial walls in the later stages of hypertension, are probably the result of persistent hypertonia. Emotional hypertension, he adds, may be regarded as a precursor to vascular disease with arterial hypertension. The treatment advocated by Stieglitz consists of avoidance of emotional stimuli and the administration of bismuth subnitrate.

Stieglitz makes the error of regarding persistent high arterial pressure as a disease rather than a manifestation of disease. The greater stability of the vasomotor system following treatment is probably due to improvement in the underlying psychical disturbance rather than to the organic effects of drugs. There is a probability also that the symptoms attributed by Stieglitz to hypertension might be due largely to emotional instability. Too much significance is apt to be attached to high blood pressure and far too many attempts, most of them quite futile, are made at its reduction. The condition of the vessel wall and the efficiency of kidneys and cardiac musculature are of paramount importance and are often neglected.

[1] *The American Journal of the Medical Sciences*, June, 1930.

THE EMPTYING OF THE GALL BLADDER.

IT is known that after a fatty meal a large proportion of the contents of the normal gall bladder is expelled. George T. Murphy has recently carried out some work at the Mayo Clinic in order to determine the effect of cholecystitis on this emptying.[1] He produced cholecystitis in ten dogs by giving them intravenous injections of eusol. The animals were made to fast for three or four days, they were then given a meal of egg yolk and cream and four hours later the abdomen was explored. The lesions were observed and three animals in one group were given intravenous injections of rose bengal. The duodenum in each of these was opened so that discharge of the dye from the gall bladder might be observed. The contents of the gall bladder were aspirated and an equal quantity of iodized oil was injected. Radiograms were taken before and after a second fat meal. In another group of three dogs the procedure was the same, with the exception that the dye was not injected and the duodenum was not opened. Four dogs were treated like the second group, except that the fat meal was not given prior to operation and radiograms were not taken until twenty-four hours after operation. One dog was used as a control. In only one instance were any signs of emptying found. Murphy concludes that the indication is that the acutely inflamed gall bladder does not empty after the usual test meal. The work is full of interest, but the conclusion must be accepted with caution on account of the artificial nature of all the circumstances.

[1] *Archives of Surgery*, August, 1930.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Intracapsular Extraction of Cataract.

K. SAFAR (*American Journal of Ophthalmology*, March, 1930) describes the method employed by him in the intracapsular extraction of cataract. The pupil is dilated with homatropine and akinesia of the lids is produced by "Novocain" injected along the inner half of the orbital margin. Cocaine is instilled and a "Novocain" injection is made above deep into the orbit. A bridle suture is then inserted through the tendon of the superior rectus muscle close to its insertion. The upper lid is held up with a Desmarres lid-holder by the assitant's right hand, the thumb of his left hand holding the lower lid down. In making the incision hæmorrhage should be avoided. After the incision is made the sutures are inserted into the conjunctival flap and the basal iridectomy is performed. To extract the lens, the blunt capsule forceps are made to seize the capsule in the lower part of the pupil, it being necessary at the same time to exert some pressure against the globe with a tenotomy hook below the limbus. Under somewhat greater counter pressure with the end of the strabismus hook, the lens, grasped by its anterior capsule, is slightly raised with its lower margin forward, so that the lower part of the zonule is first stretched and broken. The zonule is next ruptured laterally by movements to right and left until the lens follows the capsule forceps without resistance. Then the lens, with further elevation of its lower part, is drawn for the first time over the lower pupillary margin forwards and upwards, followed by pressure with the strabismus hook. Theoretically the lens should have to turn 180° upon its transverse axis if it were entirely rigid, but it clings to the pupil and the incision in such a way that it usually comes out deformed, as when one seizes and plucks an overripe fig. The zonula fibres tend to rupture last above. The iris is replaced, the suture tied, eserine is instilled in drops and ointment, and the eye is bandaged.

Ignipuncture in Detachment of the Retina.

H. S. GRADLE (*American Journal of Ophthalmology*, April, 1930) contests Gonin's claim that retinal detachment follows the formation of a hole or tear in the retina. It might be argued that as a result of exudative chorioiditis the retina is lifted from its position; subsequently the toxins of stasis have a lytic effect on the tissues and the retinal tear results. Gonin states that a cure cannot be expected if the retinal rent is not included in the cautery scar, but a successful result has been reported after cauterization when no rent could be found. The author has performed the operation five times upon three patients. In the first case, one of long standing bilateral detachment, only anatomical results could be expected. In the second case two retinal rents were present and two ignipunctures were performed; in each instance the edges of the rent were included in the scar. The detachment, however, progressed and became complete. In the third case the detachment was only ten days old and the rent could be seen. An ignipuncture, involving the rent, was performed, but with no good results. Ten days later a second puncture was made after location of another rent, but still with no benefit. In spite of his experience, the author still intends to recommend the operation as offering a better chance of effecting improvement than any other method.

Obscure Sympathetic Phenomena.

UNDER the caption "Obscure Sympathetic Phenomena" G. L. Johnson (*Archives of Ophthalmology*, July, 1930) reports two cases. The first patient, a man forty-two years of age, had had his left eye removed two years previously for an ocular tumour and had had a glass ball inserted in Tenon's capsule. His remaining eye was causing him trouble, being subject to recurring inflammation. Some ciliary infection was present. He complained of difficulty in reading, his sight "came and went." He was deceived in regard to the distance and position of near objects. The edges of letters appeared blurred. On removal of the glass ball all these symptoms disappeared. The second case was one of so-called sympathetic keratitis. The patient was a woman, aged thirty-nine years, who had suffered for five years from corneal nebulæ and iritis in both eyes. It was decided to perform iridectomy in each eye. The iridectomy in the right eye presented great difficulty owing to the adhesions of the free edge of the eyelid to the lens capsule, but it was eventually accomplished. Both eyes were bandaged. Four days later the author was surprised to find that the nebulous condition had almost entirely disappeared from the left eye.

Conservation of the Lachrymal Sac.

F. N. KNAPP (*American Journal of Ophthalmology*, June, 1930) advocates the dacryo-cysto-rhinostomy of Dupuy-Dutemps and Bourguet and describes the technique. The skin incision begins at the level of and three millimetres mesial to the internal palpebral ligament. The first half of the incision is vertical, it then curves outwards below the infraorbital margin. The mesial half of the sac is separated from the fossa and held outwards by a retractor. The periosteum is separated from the bone and the lachrymal crest is then carefully removed with a chisel; this exposes the nasal mucosa which is at once elevated in all directions. The bone bounding the lachrymal fossa is entirely removed from the anterior to the posterior crest and from the ligament above to the canal below. This is best done with a Citelli forceps. The author prefers a horizontal incision in the sac and mucosa to the usual vertical one. The corresponding edges of the incision are united with fine catgut or silk, the Yankouer nasal needle being used. The last anterior suture is brought through the deep fascia and muscle and tied. The skin is then sutured.

The Ætiology of Lachrymal Disorders.

J. I. KEMLER (*American Journal of Ophthalmology*, July, 1930) comments upon the fact that while much has been written about the treatment of lachrymal disorders, little attention has been paid to their ætiology. In addition to the usual causes assigned for epiphora, he is convinced that stenosis of the duct, which almost always precedes dacryocystitis, may be caused by nasal polypi, disease of the accessory sinuses, scrofulous changes in the nasal mucous membrane, small abscesses in the submucosa of the nasal duct, acute rhinitis and syphilis of the nasal bones. In short, stenosis follows acute or chronic disease of the nose. The author has made a practice of searching for the foci of infection and of treating them, and has found that the lachrymal disorder clears up quickly. He reports eight cases in which there were found chronic ethmoiditis, infected teeth, pus in the antrum, chronic pansinusitis or infected tonsils.

Lipæmia Retinalis.

W. R. PARKER AND H. M. CULLER (*American Journal of Ophthalmology*, July, 1930) review thirty-six published cases of *lipæmia retinalis* and discuss two of their own. The first patient was a young farmer, aged twenty, who gave a history of having been unconscious for three days and of having undergone previous treatment with "Insulin" and diet. He was placed on a high fat diet without "Insulin" and was discharged. Eight months later he returned very ill with glycosuria and acetonuria and *lipæmia retinalis*. The disc was greyish in colour, the vessels were engorged and the arterial reflex stripe was increased. Near the disc the arteries were salmon pink in the centre, paling to a cream colour near the walls. The veins were lavender coloured and further from the disc they were ivory white. The blood contained 11·6% total lipoids. With treatment the glycosuria cleared up and the fundi became normal in about four weeks, the total lipoids being 2·26%. The patient was kept on a high fat diet and this is regarded as disproving the theory that *lipæmia retinalis* is produced by fat in the diet. For twenty-seven days the patient was allowed to have a gradually increasing glycosuria without acidosis and without changes in the fundi. He was finally allowed to develop an acidosis on the fifth day; he then manifested *lipæmia retinalis* which disappeared rapidly when

"Insulin" was again given. From a review of the literature it is shown that *lipæmia retinalis* occurs typically in young diabetics; the neuroretinitis is largely confined to older diabetics. Every patient reported in the literature except one had diabetes and probably acidosis.

LARYNGOLOGY AND OTOLOGY.

A Worm in the Apophysis Mastoideum.

STEINMAN (*The Journal of Laryngology and Otology*, July, 1930) records the finding of a living ascaris which had made its way into the *antrum mastoideum* and the terminal cell of the mastoid process. The patient was a girl of eight years who had an acute mastoiditis following scarlet fever. Four days before operation the author examined the patient and could not detect anything abnormal in either ear. On opening the antrum and terminal cell of the mastoid he discovered a living ascaris surrounded by pus. He is of the opinion that the ascaris had made its way into the middle ear shortly before operation, having migrated from the intestine into the stomach, from there through the œsophagus into the naso-pharynx, and from there through the Eustachian tube, the tympanal cavity and the *antrum mastoideum* to the apical cell.

Chronic Suppurative Otitis Media.

M. D. LEDERMAN (*The Laryngoscope*, June, 1930) describes his experience for the past fourteen years with the dry treatment of chronic suppurative *otitis media* with iodine powder ("Sulzberger"). The histories of a number of patients treated are given, together with a detailed description of the author's method of applying the treatment. The preparation is an intimate association of iodine with boracic acid, the same being prepared by mixing an iodine solution of desired strength with dry, finely powdered boracic acid and evaporating the solvent. The action of the powder is due to the iodine; as the boracic acid dissolves in the secretions iodine is liberated in a most minute division and then spreads over and penetrates deeply into the tissues. This powder does not cake, but is completely dissolved within forty-eight hours, depending on the quantity of secretion present in the infected ear; it therefore does not retard drainage. Irrigations to cleanse the ear are forbidden. The canal and middle ear must be cleansed as thoroughly as possible, with removal of granulations of sufficient size and diseased ossicles, if these are visibly involved. Small perforations should be enlarged so that the powder can reach the diseased area. After all secretion has been removed, the canal and middle ear are cleansed with 95% alcohol and then dried. The final procedure is to insufflate the iodine powder into the middle ear through the perforation. For initial treatment a 0·75% iodine powder is used. The author's conclusions are: (i) Chronic suppuration of the middle ear when labyrinthine irritation, severe headache and pains or facial involvement are absent, should be treated persistently before an operation on the mastoid is recommended. (ii) Suction carried out by means of a small cannula is of great value. (iii) Remedies containing iodoform have a very beneficial action on the chronic suppuration process, especially the iodized boracic acid powder. (iv) Water introduced by way of the external auditory meatus is most harmful to the chronically discharging ears. (v) All contributing pathological changes, whether local, as granulations and polypi, or as nasal and pharyngeal disease, as well as all general conditions should have attention.

Otosclerosis: A Metabolic Disorder.

LOUIS MIRVISH (*The Journal of Laryngology and Otology*, July, 1930) presents an intensive study of three patients suffering from otosclerosis who were treated with parathormone injections and whose hearing was continuously controlled by quantitative hearing tests. The three patients were under observation for from one to three years. The author concludes that parathormone administration definitely arrested the progress of the deafness and in two patients produced considerable improvement in hearing. The improvement occurred within the first two months of treatment, but beyond that point no further improvement took place, even though the parathormone dosage was increased. On the other hand, the improvement has been maintained all the time. It is suggested that otosclerosis is analogous to osteomalacia and rickets and that these three metabolic diseases have as the basis of their pathology a common factor, namely, a state of hypoparathyreoidism.

Upper Respiratory Infection and Pulmonary Diseases.

I. RUEBERT SMITH (*The Journal of Laryngology and Otology*, April, 1930) in a paper dealing with upper respiratory infection in its relation to pulmonary disease gives a short account of his findings as oto-laryngologist to a large chest clinic. Over a period of two years he examined 1,510 patients suffering from serious chest trouble. Of these 166 proved to be tuberculous. Of the remaining 1,344 the major portion was diagnosed as suffering from bronchitis, subacute bronchitis, chronic bronchitis, bronchiectasis and asthma. In a large number of patients the lesion was found more commonly in the base of the right lung, which is explained by the direct drainage of infective secretions of the nose and throat down the right bronchus to the right lung. When nasal obstruction was present, the patient could not breathe properly and deeply, and the apices of the lung were involved on account of deficient expansion of the upper lobes. The outstanding features of these chest conditions, secondary to nose and throat conditions, are the apparent chronicity, the lack of calcareous deposits in the lung tissue, involvement of the hilar glands, the rapidity with which involvement of the glands clears up under proper treatment and the little residue left after resolution has occurred. Nasal sepsis can affect the lungs or lower respiratory tract in three ways: By direct extension of the inflammation to the larynx, the trachea and bronchi; or by infection through the blood or lymphatic stream; by aggravating or increasing an existing lesion of the lungs. The nasal sepsis may be part of general infection in which both the upper and lower respiratory tracts are involved. Of the 1,344 non-tuberculous patients 464 had nasal obstruction of varying degree; 582 patients had sinus involvement or postnasal discharge of varying character, 76 had various types of ulceration of the nasal mucous membrane. The remaining number had the following conditions; diseased tonsils, enlarged tonsils and adenoids, elongated uvula, increase of hypertrophied tissue of the pharynx, pyorrhœa, caries, granular pharynx and various conditions of the lungs. No condition of the chest should be finally diagnosed without a thorough search for foci of infection in the head. The patient's history of absence of nasal trouble should not be accepted alone without investigation of the sinuses. No diagnosis should ever be finally made on the evidence of X ray plates of the chest and sinuses.

Naso-Pharyngeal Fibroma.

HASSAN BEY SHAHEEN (*The Journal of Laryngology and Otology*, April, 1930) publishes a paper based on the experience of fifty-eight cases of naso-pharyngeal fibroma. The greater number of these growths are from the basioccipital and basisphenoid bones. The smallest growth in his series was in a child of two years; it was the size of a grape. The largest was the size of a man's fist with prolongations into the sphenomaxillary fossa. Small growths are easily taken out through the external nares. The basal attachment is severed with a pair of scissors and the growth wrenched out with a pair of Luc's forceps. When the growth is of large size, a Moure's lateral rhinotomy incision gives the best access. The base of the tumour is first attacked from the mouth. A big pair of blunt-nosed scissors curved on the flat is used to snip off the growth from the basal aponeurosis. The blunt end of the scissors is used as a wedge to lift up the tumour from its attachment. The anterior part of the growth can then be separated by means of an ordinary periosteal elevator and a good grip of the growth having been taken with a strong pair of forceps, it is pulled out. When the growth is completely out there is usually a gush of blood, but bleeding soon stops. Of the patients in this series four died.

Special Abstract.

BRIGHT'S DISEASE.

AN important study has recently been made for the Medical Research Council of the Privy Council by Dr. Dorothy S. Russell.[1] Reference is made elsewhere to the significance of this work as far as tests of renal efficiency are concerned. An attempt has been made herein to gather together the essentials of Dr. Russell's study and it is suggested that this summary be read in conjunction with the articles by Dr. Hansman, Dr. Holmes à Court and Dr. Earlam published in this issue. It should be pointed out that Dr. Russell's work practically bears the imprimatur of Professor Hubert M. Turnbull, of the London Hospital.

It is stated in the introduction that Dr. Russell's attempt has been to supply working principles whereby the physician may pass from the ascertained clinical facts to an interpretation of the underlying structural changes, of their natural history and of their probable results.

Bright's disease is defined provisionally as an idiopathic non-suppurative inflammation of the kidneys. Those forms of nephritis that are clearly symptomatic, are treated separately. It is admitted that this definition of Bright's disease is artificial and serves as a temporary guide until the ætiology of the disease has been worked out. The symptomatic inflammations of the kidney comprise those which complicate bacterial infections, lardaceous disease and various exogenous and endogenous poisons. The primary arteriosclerotic kidney should be included in the symptomatic group. The structural changes of the kidney, characteristic of arteriosclerotic atrophy, are, however, very intimately associated with the more chronic inflammations of Bright's disease. Dr. Russell has therefore included arteriosclerotic kidney as a form of Bright's disease in order that its relationship to the other forms may be fully examined.

Histological Changes.

Dr. Russell gives a short description of the results of her observations on the normal structure of the glomerulus and she points out that there is in the literature no agreement concerning many details of its structure. She then describes the histological changes as they are found successively in the glomerulus, the tubules, the interstitial tissue and the vessels. The most important part of this section is that leading to the conclusion that there are two distinct processes leading to glomerular destruction: an ischæmic process, characterized by sclerotic thickening of Bowman's capsule, and a toxic process, characterized by proliferative glomerulitis, proliferative capsulitis, focal necrosis and adhesions.

In ischæmic glomerular atrophy the primary change is thickening of the basement membrane. There then takes place the formation of a fibrous demilune internal to the basement membrane and while this is occurring, the collagen fibrils of the stroma of the hilum of the tuft become thickened. When the demilune of fibrous tissue is fully formed, the glomerulus as a whole begins to shrink. Shrinkage advances uniformly with sclerosis of the demilune. The endothelial cells of the tuft gradually disappear and the capillary lumina collapse. The tuft is affected diffusely by this change, but the capillaries near the hilum remain patent until atrophy is very far advanced. Small amorphous particles of fat are commonly seen scattered throughout the stroma of the degenerating tuft. (It should be pointed out that Dr. Russell uses the term fat to designate neutral fats and fatty acids and the term fatty degeneration to designate the process which leads to their appearance in tissue; lipoids forming doubly refractive fluid crystals she terms lipoids and describes the process leading to their appearance in tissue as lipoid degeneration or infiltration.) The final stage of ischæmic glomerular atrophy is one of distortion and disintegration.

Toxic glomerular atrophy, as already mentioned, consists in proliferative glomerulitis, proliferative capsulitis, focal necrosis and adhesions. There are two main types. One is characterized by great proliferative activity of the epithelial and endothelial cells of the glomerulus and is associated with a brisk inflammatory reaction in the kidney; the other is characterized by glomerular changes of a degenerative nature and is associated with a milder reaction in the remainder of the kidney. There is no sharp division between these types.

It is thus evident that the two classes of change, the ischæmic and the toxic, are the result of two fundamentally different causes: the ischæmic is the result of partial interference with the blood supply and the toxic is the result of bacterial or other toxins.

The Nature of Ischæmic Nephritis.

The chief evidence of the nature of ischæmic nephritis is afforded by its close association with chronic arterial degeneration in the absence of any other pathological condition in the kidney. This arterial degeneration may or may not be a complication of cardio-vascular hypertrophy.

It is extremely common to find on microscopical examination of apparently healthy kidneys in adults ischæmic atrophy of a few glomeruli and associated tubules. Dr. Russell found that in subjects over thirty years of age the kidneys were very rarely normal. Some amount of ischæmic atrophy was almost invariably present among the younger subjects and became constant with advancing years. The presence and number of the atrophied areas corresponded closely with the presence and degree of arterial degeneration.

In almost all the cases of cardiac hypertrophy examined by Dr. Russell some amount of ischæmic nephritis was detected in the kidneys. The amount was usually greater, the greater the degree of cardio-vascular hypertrophy; but this was true only in so far as the arterial degeneration tended to be greater, the greater the cardio-vascular hypertrophy. There was a much closer association between the amount of ischæmic nephritis and the amount of arterial degeneration, in particular intimal degeneration.

The type of nephritis that is found closely associated with chronic degeneration of arteries, is also found in association with compression of the renal cortex by hydronephrosis, tumours and cysts. When this nephritis is associated with vascular degeneration, it has been shown that there can be little doubt that it is due to interference with the blood supply. Dr. Russell regards it as reasonable to assume that gradually increasing compression would also interfere with the blood supply and would lead to ischæmic changes.

The Nature of Toxic Nephritis.

The ischæmia of ischæmic nephritis differs from that which causes a typical infarct, that is to say, an area of coagulation necrosis, in that it is less rapid and less complete; it is a chronic ischæmia as opposed to the acute ischæmia of ordinary infarction. Although in Bright's disease arterial degenerations may be present in kidneys which are the seat of toxic nephritis, yet they are frequently absent. Where such arterial degenerations are present, the amount and distribution of the degenerations bear no relation to the amount and distribution of the toxic nephritis. The toxic nephritis in Bright's disease is clearly not due to either acute or chronic ischæmia. To distinguish it from the nephritis of Bright's disease which is due to chronic ischæmia, Dr. Russell suggests that it might be called an-ischæmic. Not only, however, do the inflammatory reactions bear a general resemblance to those which are caused in other tissues by bacterial and other toxins, but there is evidence that essentially similar or identical forms of nephritis are actually caused by such toxins. This evidence is set out by Dr. Russell in a chapter in which she deals with histological types of kidneys in symptomatic nephritis. She regards the use of the term toxic as amply justified.

[1] "A Classification of Bright's Disease," by Dorothy S. Russell, Medical Research Council of the Privy Council, Special Report Series, Number 142.

The Combination of Ischæmic Nephritis with Toxic Nephritis in Bright's Disease.

In a large number of kidneys a combination of ischæmic and toxic nephritis is present. There is no evidence that ischæmic nephritis tends to be complicated in its later stages by a toxic nephritis. On the other hand, toxic nephritis is usually found to be pure only in its early stages. There is a manifest tendency for ischæmic changes in the cortex to become increasingly conspicuous as the toxic nephritis proceeds. There is no evidence that ischæmic nephritis predisposes the kidney to toxic nephritis. The tendency for cardio-vascular hypertrophy with arterial degeneration and consequent ischæmic nephritis to occur and to increase in the later stages of prolonged toxic nephritis accounts for the ischæmic nephritis in all save a small proportion of kidneys in which a toxic nephritis is combined with an ischæmic nephritis of abnormal amount. In this small proportion the vascular changes and ischæmic nephritis seem to have preceded the toxic nephritis. This in Dr. Russell's opinion does not modify the conclusions already stated. It is inevitable that such cases should be found in the survey of any large amount of material, because the pathological combination of cardio-vascular hypertrophy and primary ischæmic nephritis is so commonly met with in *post mortem* examinations. Unless it could be demonstrated that the existence of these conditions conferred immunity to toxic nephritis, kidneys are certain to be found in which an unusually large amount of ischæmic nephritis has preceded the toxic.

Histological Classification of Bright's Disease.

Dr. Russell makes a classification of Bright's disease according to histological criteria. From what has been written, much of this classification will be obvious. Clarity will best be served by setting out the various groups before entering into further detail. The classification is as follows:

I. Primary ischæmic nephritis (arterio-sclerotic kidney).
   1. Due to vascular degeneration without cardio-vascular hypertrophy.
   2. Due to vascular degeneration complicating cardio-vascular hypertrophy (kidney of essential hypertension).

II. Toxic nephritis, with secondary cardio-vascular hypertrophy and ischæmic nephritis in late stages.
   A. Low grade or mild toxic nephritis.
      1. Mild nephritis. *Nephritis mitis.* (Genuine nephrosis of Volhard and Fahr. Lipoid nephrosis of Munk. One form of parenchymatous nephritis of most text books.)
         a. In early stage.
         b. In intermediate or granulation stage.
         c. In chronic or sclerotic stage.
   B. High grade or intense toxic nephritis.
      1. Intense nephritis. *Nephritis acris.* (Commoner form of parenchymatous nephritis of most text books. Glomerulo-tubular nephritis.)
         a. In early stage.
         b. In intermediate stage.
         c. In chronic stage. (Two chronic diffuse types.)
      2. Creeping or insidious (intense) nephritis. *Nephritis (acris) repens* (chronic interstitial nephritis).
         a. In early stage.
         b. In intermediate stage.
         c. In chronic stage. (Four reticular types.)

III. Toxic nephritis complicating primary ischæmic nephritis.

It will be noted that toxic nephritis is divided into two distinct classes. Dr. Russell claims that these two classes of inflammation are probably the result of two distinct classes of toxin. Class *A*, *nephritis mitis*, is characterized by low grade active inflammatory reaction. Proliferative glomerular changes are absent and cellular infiltration is slow to appear, scanty and of low grade. The parenchymatous change is characterized by the absence of hyaline droplet degeneration and by the magnitude of lipoid infiltration in the initial stage. This kind of inflammation is closely allied to that known to be caused by certain endogenous poisons. In the kidneys investigated by Dr. Russell no example of nephritis complicating bacterial infection was of this low grade.

The nephritis of Class *B* is characterized by the intensity of active inflammatory reaction. Proliferative glomerular changes are associated with a cellular infiltration, both abundant and rich in neutrophile leucocytes. Albuminous and hyaline droplet degenerations preponderate in the initial parenchymatous change. When bacterial infections other than the mildest in Dr. Russell's series were complicated by nephritis, the nephritis was undoubtedly of this kind. The nephritis complicating the mildest bacterial infections were more akin to Class *B* than to Class *A*.

The form of nephritis characterized by low grade of inflammatory reaction has been recognized by Volhard and Fahr, and by Munk, and has been called simple nephrosis and lipoid nephrosis respectively. It is in Dr. Russell's opinion correct to apply the term nephrosis to any specimen of this renal condition in which parenchymatous degeneration alone is present, but it does not appear justifiable to apply the term nephrosis to the process as a whole. It is incorrect to speak of the inflammatory stages of a nephrotic process.

The inflammation of peculiarly mild intensity or low grade certainly requires some designation to distinguish it from the intense inflammation of high grade (Class *B*). The terms acute, subacute and chronic are used to express time relations as well as intensities and according to Dr. Russell's findings the more intense inflammations can persist for an even longer time than the mild. Moreover, the use of these qualifications would not harmonize with their use in many clinical classifications of nephritis. The terms low grade or mild nephritis, *nephritis mitis*, and hgh grade or intense nephritis, *nephritis acris*, express the respective histological characteristics. *Nephritis acris* may be either widespread in its initial involvement of the kidney, leading to a relatively rapid destruction, or may be focal initially and lead to a slow, apparently intermittent form of destruction. These differences call for special nomenclature. The form characterized by initial limitation and slow progress of inflammation, is distinguished by Dr. Russell as insidious nephritis, *nephritis acris repens*, or for short, *nephritis repens*. If the old names are retained, the term arteriosclerotic kidney should be confined to kidneys showing primary ischæmic nephritis. *Nephritis mitis* in all stages and *nephritis acris* of extensive initial incidence would correspond to parenchymatous nephritis. *Nephritis acris repens* would correspond to chronic interstitial nephritis.

Dr. Russell discusses in detail the types of chronic toxic nephritis—the two chronic diffuse types and the four chronic reticular types. Limitations of space prevent detailed description of them. It must suffice to point out that she believes the six types to be connected histologically. Their peculiarities depend essentially upon variations in the intensity of a similar inflammatory reaction, variations in the initial incidence of this inflammation and in the rapidity of its extension. These three factors do not vary in corresponding degrees.

Clinical Manifestations.

The clinical information in Dr. Russell's series was obtained by the ordinary clinical notes and by the results of specific tests for the estimation of renal efficiency. The tests employed were the estimation of the blood urea, the estimation of phenolsulphonephthalein excretion after intramuscular injection of the dye, and Maclean's concentration test.

In primary ischæmic nephritis no clinical evidence was found apart from the occasional excretion of a trace of albumin; all abnormalities in the results of the tests for renal sufficiency complicated heart failure and were such as are known to be caused by heart failure. The cause of death was determined in thirty-six instances. In eleven

the cause was entirely unrelated to the cardio-vascular and renal systems; heart failure was responsible for death in nineteen instances and cerebral hæmorrhage in six.

The early and intermediate stages of *nephritis mitis* are similar in that the patients affected are usually children or young adults who appear to be free from any predisposing cause. The disturbance is characterized by an insidious progressive œdema accompanied by relatively little constitutional disturbance and by the absence of cardio-vascular hypertrophy. There is difficulty in eliminating water by the kidneys, but no difficulty in excreting nitrogenous products and phenolsulphonephthalein. The clinical features are those of an insidious chronic disease. The disease does not appear to be fatal in itself; the danger to life lies in the liability to contract a concurrent pyogenic infection, the commonest infecting organism being the pneumococcus. The early stage differs from the later only in the relative shortness of the clinical history.

Nephritis acris is an illness principally of children or young adults. Dr. Russell found a great similarity between the clinical manifestations of all examples in the early stage and those of the more intense type in the intermediate stage. Both are characterized by profuse hæmaturia, attended by such symptoms as vomiting, headache and abdominal pain and in some instances a moderate degree of œdema. There is some evidence that the disorder is initiated by a bacterial infection. The renal disturbance is severe and appears to be the chief cause of death. The patients in Dr. Russell's series in whom the disease in its early stage was fatal, were mostly children, whereas in the intermediate stage the disease was confined to adults. The less intense type of *nephritis acris* in the intermediate stage is in some respects different from the intense type. The onset is more insidious and œdema is usually a more conspicuous clinical feature. Hæmaturia, although present, is never great, and the disturbance of nitrogen excretion is never so evident as in the more intense type. The average length of the history is considerably greater and there is a tendency towards the concurrent development of cardio-vascular hypertrophy. When death occurs in this type, it is less often due to renal inefficiency than to accessory factors, such as œdema of the lungs and secondary infections of the serous sacs. The patients in this type may survive until the late or chronic stage has developed. The chronic stage of *nephritis acris* is distinguished by the addition of constant cardio-vascular hypertrophy, albuminuric retinitis, partial or complete loss of œdema and by the development of typical uræmic symptoms. There is chemical evidence of gross renal insufficiency in this stage and this in all cases appears to be the cause of death.

Nephritis repens which has already been mentioned as occurring in four types, is sometimes preceded at a long interval of time by an attack similar to that of the early stage of *nephritis acris*, but usually the terminal stage of uræmia is preceded only by trivial symptoms. With the exception of the third type in which children and young adults are affected and renal regeneration is maximal, cardio-vascular hypertrophy and consequent ischæmic nephritis are prominent features and are greatest in the most chronic type.

In regard to toxic nephritis complicating primary ischæmic nephritis, it is impossible to make a clinical differentiation of this condition from primary *nephritis acris* in an intermediate or late stage or from primary *nephritis repens*.

Clinical Nomenclature.

The nomenclature used throughout the report of the investigation is based on histological features. Dr. Russell concludes that when such a name is used at the bedside, there is a danger of the name being taken to imply a clinical feature. *Nephritis mitis* is not a mild disease; it is as serious and lethal as that in which the inflammatory reaction within the kidney is intense. Names that express phenomena that can be observed, can be used with greater security. Dr. Russell therefore suggests the following nomenclature. Primary arteriosclerotic nephritis is more appropriate clinically than primary ischæmic nephritis. *Nephritis acris* is clinically an acute nephritis. *Nephritis mitis* is characterized by œdema without retention of nitrogen in the blood. This has been distinguished in the past by the term hydropigenous or more incorrectly hydræmic. *Nephritis mitis* thus becomes subacute hydropigenous nephritis. In the intermediate stage of *nephritis acris* there is retention of nitrogen. The intermediate and chronic stages of *nephritis acris* therefore become subacute and chronic azotæmic nephritis. *Nephritis repens* is then to be known as insidious chronic azotæmic nephritis.

Special Articles on Diagnosis.

Contributed by Request.

XIII.

PYELITIS AND KIDNEY PAIN.

"PYELITIS" is a very popular diagnosis when a patient presents himself for treatment suffering with pain, discomfort or tenderness in one or other kidney region, associated with the presence of pus in the urine and perhaps frequent and painful micturition and more or less elevation of temperature.

"Pyelitis," that is to say, a more or less superficial inflammatory lesion involving the renal pelvis and possibly calyces, is, nevertheless, an extremely uncommon condition as a disease entity. While it is a common assumption that mild cases do occur resembling somewhat in their incidence a common cold, it is an undoubted fact that the majority of patients with these symptoms are suffering from a much more tangible lesion.

It will be of no particular value to give a statistical estimate from one's own practice of the various lesions popularly labelled as "pyelitis," because so many of the mild, self-limited types which are so common in general practice, do not reach the urologist.

In a consecutive series of one hundred and seventy patients suffering from proved renal pain which I recorded in THE MEDICAL JOURNAL OF AUSTRALIA in January, 1919, only fifty-one, that is, 30%, had urinary calculi, and this is in accord with the general experience.

From this it will be apparent that in more than two-thirds of the patients who are suffering from renal pain, the plain X ray examination of the urinary tract for stone (combined even with microscopical examination of the urine) will be insufficient to make an accurate diagnosis.

Renal tuberculosis has been previously considered at length in these articles and will not be specifically alluded to here.

The repeated attacks of so-called "pyelitis" of a more or less severe grade to which so many people are prone, are due not uncommonly to localized septic infarction of the kidney of hæmatogenous origin. These lesions are generally bilateral, readily spread beyond the capsule and cause renal pain frequently on only one side. Surgical operation, except in fulminating or persistent acute cases, should be resorted to only when the focus of infection has been carefully sought for and eliminated. This disease in its milder form is most commonly found as an accompaniment of infection of the upper respiratory passage—tonsils, antrum *et cetera*—and occasionally of the appendix or uterine adnexa, and is typically associated with the *Bacillus coli communis*, while the severer grades are more apt to be associated with a coccal infection and to follow some acute localized septic lesion, such as furunculosis, quinsy *et cetera*.

Renal pain, however, in the vast majority of cases, is a reflex of increased intrapelvic pressure, due to some form of obstruction to the free flow of urine through the urinary excretory apparatus. The stasis of urine which results, leads sooner or later to infection. It is the onset of this infection or its repeated lighting up which induces the attacks of so-called "pyelitis."

"Pyelitis," then, is merely a symptom or indication of disease and not a diagnosis. The futility will be evident, therefore, of treating these patients either by prolonged courses of vaccine or by bladder irrigation because of the accompanying frequency and pain which is so commonly misdiagnosed as cystitis. This type of so-called conservative treatment has long been, and unfortunately still is, widely exploited.

Urinary stasis may be caused by obstruction within the urinary tract itself, that is to say, of intrinsic type, or by pressure from without, that is, of extrinsic type.

Intrinsic Causes of Urinary Obstruction.

The intrinsic obstructions may occur anywhere from the calyces to the orifice of the urethra. Amongst the commonest are stricture of the ureter, enlargement of the prostate, carcinoma of the bladder and in children and infants various types of congenital obstruction at the bladder neck and prostatic urethra.

There is another type of intrinsic obstruction due to disturbances in the neuro-muscular apparatus of the kidney itself which is just gaining recognition. This comprises a group of phenomena or syndrome to which the writer has given the title of "renal sympathetico-tonus." Briefly, the renal pain which is characteristic of this group of cases, is due to overaction of the sympathetic nerve supply of the kidney. Spasticity of the muscles, with incoordination of the contractions of the calyces, pelvis and ureter, is thus induced with resulting increased intracalycal and intrapelvic tension.

Renal sympathetico-tonus is a definite clinical entity and is curable by division of the nerve supply to the kidney, the so-called "renal denervation" or "renal sympathectomy."

We have found that renal sympathetico-tonus is often, if not always, combined with generalized sympathetico-tonus, sometimes actually localized to the side of the renal lesion, as manifested by increased excitability of the knee jerks and delayed relaxation time.

This condition of renal sympathetico-tonus is a very important one and will at no distant date come to be recognized as playing an important part in the ætiology of many painful states of the kidney, including some types of hydronephrosis.

A considerable amount of research has been carried out on this subject at the Urological Department of the Lewisham Hospital and at the Medical School of the University of Sydney by my brother, Dr. Richard Harris, and myself during the past four years, the results of which have been embodied in a paper presented at the symposium on sympathetic surgery at the British Medical Association, ninety-eighth annual meeting, held at Winnipeg in August, 1930.

Extrinsic Causes.

The commonest of extrinsic causes are obstructive bands, kinks or aberrant vessels at the uretero-pelvic junction and upper third of the ureter, or obstruction in the lower half of the ureter by pressure of the pregnant uterus, malignant disease of the cervix or by inflammatory disease of the seminal vesicle.

The obstructions at the uretero-pelvic junction are a very common, probably the commonest, cause of hydronephrosis, and quite frequently an advanced grade of hydronephrosis is present before infection ("pyelitis") supervenes. The importance, therefore, of recognizing this condition before the kidney has gone on to complete destruction is self-evident and can be made with certainty only by complete urological investigation, that is to say, cystoscopy, pyeloscopy, pyelography *et cetera*. The recently introduced descending method of pyelography by means of the intravenous injection of "Uroselectan" promises to be of some service, but has not so far measured up to expectations.

This type of lesion is commonest in the second and third decades of life and is typically manifested by infrequent attacks of very severe pain, generally fixed in the affected loin and not radiating downwards.

When infection is superadded, any grade of "pyelitis" (pyelonephritis, pyonephrosis *et cetera*) may supervene. Between acute attacks some more or less intermittent renal aching and soreness or tenderness, with pyuria and perhaps frequency of micturition, may be the only subjective symptoms. Frequent and painful micturition does not occur in cases of simple hydronephrosis unless infection is present.

Pyelitis Gravidarum.

"*Pyelitis gravidarum*" is a condition that is very amenable to treatment and deserves a few words to itself.

The disease has its origin in some obstruction in the ureter due in some way to pregnancy. It can often be recognized in the prepyelitic stage when careful prematernity methods are employed. Pain in the right loin, with perhaps slight tenderness, being the earliest symptom.

The lesion, at any rate in its early stages, is confined practically to the right side, though at a later stage the left kidney may also be involved. When the left kidney alone is attacked, the disease is almost certainly not "*pyelitis gravidarum*" and some other cause should be sought.

My experience with a very large number of these cases during the past twenty years leads me to the opinion that induction of labour should rarely or never be necessary. Severe infections respond readily to the inlying ureteral catheter, especially when a large size, such as ten or twelve French, is employed.

More recently researches on the nerve supply of the kidney and ureter which Dr. Richard Harris and the writer have been carrying out, and the physiological work of Dr. Leon Jona, of Melbourne, have suggested the value of eserine in this type of condition.

We recommend injections of 1·08 milligrammes (one-sixtieth of a grain) of eserine hypodermically three times a day for two or three days with the patient turned on the side away from the lesion.

We have found that eserine will not cause efficient contractions of the renal pelvis and ureter in the presence of organic obstruction. Turning the patient on the side away from the lesion enables the pelvis and ureter to contract readily under the influence of eserine.

Experience to date suggests that there is a fair prospect that eserine may prove a specific for *pyelitis gravidarum*.

Conclusions.

Patients suffering from the so-called "pyelitis," whether children or adults, which does not rapidly react to simple methods of treatment, or which recurs at intervals, should forthwith be submitted to complete urological investigation.

Failure to recognize this seemingly self-evident fact is the reason for too many patients presenting themselves for urological investigation at a time when any possibility of conservative treatment is long past.

Treatment without complete investigation of patients of this type by prolonged courses of vaccine therapy and bladder irrigations cannot be too strongly condemned.

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British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on July 31, 1930, DR. E. M. HUMPHERY, the President, in the chair.

Renal Efficiency Tests.

DR. F. S. HANSMAN read a paper entitled: "Renal Functional Tests from the Biochemical Viewpoint" (see page 415).

DR. A. W. HOLMES À COURT read a paper entitled: "The Clinical Aspect of Tests of Renal Efficiency" (see page 429).

DR. M. S. S. EARLAM read a paper entitled: "Renal Efficiency Tests and Their Surgical Application" (see page 431).

DR. H. S. HALCRO WARDLAW said that he appreciated very greatly the privilege of being able to attend the meeting of the Branch. He was interested more particularly in Dr. Hansman's paper. The determination of renal function was a difficult and complex subject. As at least four stages were concerned in the secretion of urine, it was obvious that no one test could be completely satisfactory. The urea concentration test filled many useful purposes and when combined with blood urea estimation and determination of the urea concentration factor, it gave valuable information. In regard to Dr. Hansman's limitation of water intake for thirty-six hours before the application of the urea concentration test, he agreed that some control of water was desirable. The same results could be obtained by increasing the dose of urea. The question was what water ration should be allowed. In regard to Dr. Hansman's conclusion that the attainment of a constant level of urea was indicative of renal deficiency, Dr. Wardlaw said that in the Department of Physiology at the University of Sydney the students performed the urea concentration tests on themselves. In a certain proportion the urea concentration rose and stayed at constant levels. These students were apparently healthy and he concluded that a constant high level was not always indicative of deficient kidneys. He thought that some of the high concentration results obtained by Dr. Hansman were due to his limitation of water intake.

DR. C. B. BLACKBURN expressed his gratification at having had an opportunity of hearing the papers. Dr. Hansman had referred to a case of his. This had been of exceptional interest. Dr. Blackburn wished to refer to it on account of the statement that he had recommended decapsulation of the kidneys; he did not wish it to be thought that it was a common practice of his to recommend decapsulation. This was a most unusual case in which a child with normal urine the day before an operation promptly got complete suppression after it. Five days later she was having uræmic convulsions and the only urine, about one fluid drachm, obtained from the bladder by catheter had contained no albumin. He had felt that it was a kind of reflex suppression and that any handling of the kidneys would perhaps start them going. Sir Alexander MacCormick had operated rather unwillingly and had found the kidneys looking fairly normal, but had stripped the capsules. Secretion of urine in great abundance had set in at once. Dr. Blackburn agreed with the generally accepted opinion that in nephritis decapsulation was not of much value.

Referring to Dr. Holmes à Court's paper, Dr. Blackburn said he made a practice of estimating not only the percentage of urea in the specimens obtained, but also the total urea excreted. In his experience, as a general rule, if during the first two hours more than four grammes of urea were excreted, the kidneys were reasonably efficient.

Some seven years previously he had been consulted about the advisability of allowing an elderly patient with arteriosclerosis to visit Europe, as a renal efficiency test had shown 1·3% as the highest concentration of urea. Finding that five grammes were secreted in the first two hours and that the blood urea was 33, he had consented. The patient had remained in good health till shortly before his death from cerebral hæmorrhage nine months previously, but though several further efficiency tests were made, his urea concentration had never been better than 1·4%. Shortly before his death the blood urea was forty milligrammes, so that after all those years the kidneys were still reasonably efficient. It should never be forgotten that these tests must not be regarded as tests of normality, but of whether there was enough actual tissue left to act as an efficient filter, and people with arteriosclerosis who died from such complications as cerebral hæmorrhage and heart failure, never were in fact found to have normal kidneys, although tests of their functions might have given normal figures. In basing a prognosis on a renal efficiency test consideration should be given to any other evidence obtainable and this might help in deciding whether progressive changes for the worse were likely to occur soon in the kidneys.

He recalled one patient originally treated for stone in the kidney and later for nephritis with persistent heavy albumin and many casts associated with high blood pressure. Despite satisfactory urea concentration tests, the outlook had appeared grave. This man had later visited England and returned bearing a letter from an institution there, where exhaustive investigations had been made, in which a favourable prognosis had been given, despite the heavy albumin and casts and a diastolic blood pressure of 140 millimetres of mercury and a systolic pressure of 210 millimetres. This patient had died of uræmia nine months later.

There was less vagueness about the significance of the term renal efficiency when used in reference to surgical procedures where the question of durability of the kidney was not under review, but merely whether it was likely that the patient would survive a particular operation.

PROFESSOR A. E. MILLS said that he had listened to the reading of the three papers with much interest and he congratulated the authors. He wished, however, to differ in opinion from Dr. Hansman in regard to one point. Dr. Hansman had stated that the kidneys could not be regarded as normal in function unless they were able to concentrate urea to the extent of 3%, or at any rate over 2·5%, after the administration of a test dose. Professor Mills ventured to suggest that the kidneys of all persons did not conform to such an accurate standard. Information gleaned from tests of function of other organs would assist them in the interpretation of estimations of urea concentration. Who, for example, would maintain that an abnormality in gastric secretion, such as a mild hypochlorhydria or hyperchlorhydria was evidence that the stomach was incapable of performing its work adequately and well? Many persons could live quite happily and without discomfort and yet have no free acid in the gastric juice. There was no dogmatic standard and the limits of normality were wide apart. It was wrong to demand a concentration of over 2·5% or 3% in the urine after the ingestion of fifteen grammes of urea. Maclean did not demand 3%.

Again, defects in urea concentration were not necessarily evidence of renal damage. The kidneys of many patients suffering from anæmia lost some of their concentrating power, although the organs were themselves normal in structure, and in certain diseased states, not involving the renal tissues, there was an increase in the blood urea. The latter was particularly in evidence after severe hæmorrhage, whether caused by trauma, duodenal ulcer or other extrarenal affection. There was yet another factor which merited consideration in the evaluation of the results of the test. Patients who came even to the placid precincts of Dr. Hansman's laboratory might be nervous; and many present that evening could remember keenly from their experiences prior to *viva voce* examinations what nervousness could do so far as the excretion of urine was concerned. The only way to eliminate diuresis caused by nervousness was to repeat the test.

Without doubt Dr. Blackburn was right in his contention that biochemists should estimate not only the percentage, but also the total quantity of urea excreted.

DR. EVA SHIPTON said that in regard to the preparation of the patient for the urea concentration test it was necessary for the patient to abstain from fluid for twelve to fifteen hours previous to the test. She could not see that total abstention from food was necessary and she allowed patients to have a dry breakfast previous to the test should they be so disposed. She noticed that Harrison, Chemical Pathologist at Saint Bartholomew's Hospital, allowed a dry breakfast either before or during the test. The figures given by Dr. Hansman for normal values were in her opinion far too high and the percentage of urea in the specimen previous to the dose of urea varied greatly in the normal and was of little significance, some pathologists even discarding it. The maximum concentration of urea might occur in the first, second or third hour and the rate of absorption from the stomach must affect this. Dr. Hansman rightly emphasized the absolute necessity of measuring the volume of each specimen, for though a

normal concentration might occur with diuresis due to extrarenal conditions, as in *diabetes insipidus*, this was distinctly unusual. In acute nephritis the blood urea, estimated frequently, was often of great aid in prognosis, but the urea concentration test was useless and might be actually harmful. In the interpretation of these tests it was essential that every aspect of the case be considered and not merely the laboratory findings.

DR. R. GORDON CRAIG expressed his appreciation of the papers and said that sufficient stress had not been laid on the patient's appetite as an indication of his renal efficiency. He had frequently noticed that if the kidney function were going to fail, the first indication would be given by a loss of appetite. If the patient's appetite were good, all, as a rule, was well. Even if the renal efficiency tests showed that the patient was on the border-line as far as operability was concerned, it would be safe to operate upon him if his appetite were good. There was a distinct difference in medical and surgical conditions of the kidney. When a physician used renal efficiency tests he applied them to a patient who was suffering from a pathological lesion of the kidney; the surgeon, on the other hand, had to do with a disordered function, due to a removable cause. Dr. Craig then went on to discuss the phenolsulphonephthalein test. He said that a figure of 60% to 80% was normal and one of 50% was good; with a figure of 20% over the first two hours it would be safe to operate. He had even operated on a patient whose renal concentration had become stable at 5% over a period of four weeks and whose appetite was good. In regard to the relative value of the various tests, he thought that no one test was preeminent. All those in the series were fairly parallel: if one was good, all were good. One thing which these tests had done was to make surgeons realize the importance of uræmia and the possibility of its prevention. In the last seventeen deaths after prostatectomy among one hundred and fifty patients only one had been due to uræmia; thirteen had been due to infection. Uræmia had been practically eliminated as a cause of death after prostatectomy. In conclusion Dr. Craig asked Dr. Blackburn what anæsthetic had been used in the operation on Dr. Blackburn's patient.

DR. H. A. RIDLER said that he was disappointed because no one of the speakers had mentioned the best test of renal efficiency. He referred to pregnancy and added that unfortunately its application was limited.

DR. A. H. TEBBUTT said that the biochemists either did not interest themselves in the underlying morbid histology of the conditions they investigated or they said that pathologists could not tell them anything. The pathologist, of course, rarely obtained biopsy material from the kidney, and when he did obtain the kidney in the *post mortem* room it was not of much value to the patient. The pathological classification of kidneys and also the clinical classification of renal diseases had always presented considerable difficulties and was by no means finalized. He would recommend those who were interested, to read the classification of Bright's disease by Dorothy Russell and Professor Turnbull. They drew a sharp distinction between toxic nephritis and primary ischæmic nephritis (the arteriosclerotic kidney). They were distinguished both in their origin and in their clinical course. Toxic nephritis frequently ended in uræmia, whereas ischæmic nephritis ended in death from cerebral hæmorrhage, heart failure *et cetera*, the kidney outliving the heart, cerebral vessels, *et cetera*. Professor Turnbull had elaborated the histological classification and on the whole he found that it fitted in well with his classification on clinical grounds. He considered that nephrosis was a renal disease, but would prefer the term "subacute hydropigenous nephritis" (subacute hydræmic nephritis). He distinguished sharply this condition from acute nephritis, subacute azotæmic nephritis and chronic nephritis, in which there was urea retention. These forms might pass the one into the other. Chronic nephritis was subdivided into two main types, chronic diffuse and chronic insidious (*nephritis repens*), the latter being the more common. He was of the opinion that urea concentration tests had proved useful and this was quite flattering coming from a man who was steeped in morbid anatomy. He particularly emphasized their value in distinguishing between ischæmic nephritis and chronic insidious nephritis when both showed cardiovascular hypertrophy. He thought that the old descriptive terms, large white, small white, small red granular kidney *et cetera* should be given up in favour of terms which expressed the underlying histological picture.

DR. RICHARD HARRIS said he would like to add his congratulations to the authors of the three papers for their most interesting and instructive contributions. In the experience of Dr. Harry Harris and himself in their urological department at Lewisham Hospital, the indigo-carmine test had never been found wanting as a test of surgical renal efficiency during the past twenty years.

A normal test indicated merely that prostatectomy or renal surgery could be undertaken with the assurance that the kidney would not stop work, whilst an unsatisfactory test indicated in their experience a uræmic death following major surgical interference. Those patients who yielded unsatisfactory results to the indigo-carmine test after prolonged catheter drainage, were submitted to cystotomy as a preliminary to later prostatectomy, if and when their renal function had sufficiently recovered. No prostatic patient with complete retention was refused operation unless patently moribund. Practically any patient with a dirty bladder could be got sufficiently clean to undergo a one-stage operation by persistent irrigation followed by the instillation of four ounces of a one in three thousand solution of silver nitrate, or stronger if tolerated; this was retained for half an hour. The few patients whose bladders could not for various reasons be cleaned up, were submitted to preliminary cystotomy. Silver nitrate, in their experience had proved far more satisfactory than any of the recently introduced urinary antiseptics. The dirtier the bladder, the greater was the tolerance for silver nitrate.

Thompson-Walker had well stated in his recent Lettsomian Lectures: "Sepsis in relation to the enlarged prostate and prostatectomy is a failure of surgical methods." The post-operative vesical and pelvic sepsis mentioned by some previous speakers should not occur in a properly conducted case.

With regard to the indigo-carmine test, for some considerable time they had been using eight cubic centimetres of a 0·8% indigo-carmine solution as an intravenous injection. With a 0·4% indigo-carmine solution they found the results were not uniform.

In those prostatics undergoing catheter drainage, the catheter was disconnected from the drainage bottle and allowed to run freely into a series of sterile test tubes. A blue coloration should appear within five minutes, increasing in intensity to a deep blue within ten minutes. A greenish blue was not a satisfactory test, while a pale transparent blue suggested the possibility of uræmia if the patient was operated on. They had repeatedly checked up these poor indigo-carmine tests with blood urea and blood creatinine tests, and in many cases found a normal or slightly raised blood urea and blood creatinine where a very poor indigo-carmine excretion had been recovered.

One patient, a judge, was operated on for prostatectomy in such circumstances. After prolonged preparation the renal condition was considered to be at its optimum point. He developed complete anuria for forty-eight hours and hiccup for one week and finally completely recovered. A recent patient with complete retention of urine and coronary sclerosis, whose blood urea and blood creatinine had returned to about normal after five weeks' catheter drainage and whose indigo-carmine showed a poor blue in ten minutes, was not so fortunate. They had been prepared for the possibility of a fatal issue in this case from their interpretation of the result of the indigo-carmine test and, in fact, the relatives had been so warned.

In assessing operability Dr. Harris said that the general condition of the patient was, of course, taken into consideration, but he would emphasize that, however well the patient might appear, prostatectomy was never undertaken unless the result of the indigo-carmine test was considered good enough to warrant it. Thus, while realizing the value in some cases of the various blood tests.

they were definitely opposed to unnecessary multiplication of them.

In a personal communication to his brother, Dr. Harry Harris, Dr. Frank Kidd, of London, wrote that he was in entire accord with their position relative to the indigo-carmine test. He said in effect, "with you I consider this the final and absolute test of operability."

With regard to anæsthesia, they found the excretion of indigo-carmine following intravenous injection only very slightly delayed under ether anæsthesia, and consequently raised their limits to fifteen minutes for a deep blue colour as an indication of normal renal function. They had not found the relative function of the two kidneys, whether in a normal or subnormal state, grossly interfered with by anæsthesia, except on rare occasions.

With regard to bilateral polycystic disease of the kidneys, they found that Rovsing's operation had yielded extremely satisfactory results in a large series of cases over a period of more than twenty years. Operation, however, must not be undertaken in the absence of satisfactory functional test in at least one kidney.

The work of Hinman on renal counterbalance had been very largely discounted of recent years and many kidneys which, according to his theories, should not have been worth saving, had regained useful function after conservative surgery.

A further test which had not been mentioned, was the carbon dioxide combining power of the blood plasma. The normal blood had an alkali reserve equivalent to from 53 to 70 cubic centimetres of carbon dioxide per hundred cubic centimetres of plasma. A decrease below 50% by volume indicated an alkali deficiency, that was, an acidosis, whereas an increase above 70% by volume denoted an alkalosis. This test of the carbon dioxide combining power of the plasma they considered of great importance in those surgical cases tending towards uræmia.

By the use of the duodenal tube greater quantities of fluid could be given and lavage *en masse* of the bowel could be easily performed—up to one gallon of fluid could be given.

An acidosis or an alkalosis could be combated, or easily produced, by the administration of bicarbonate of soda or glucose on the one hand, or sodium chloride on the other.

When in doubt as to the alkali reserve plain tap water should be given.

This method of introduction of great quantities of fluid and lavage *en masse* they believed to be the best stimulant to kidney function at their disposal in cases of surgical renal failure.

A method of estimation of the indigo-carmine retained in the blood in relation to the amount excreted in the urine had recently been introduced.

This offered great possibilities and would particularly interest their biochemical friends. It permitted a quantitative estimation of the indigo-carmine blood retention. Of this they hoped to say more at a later date.

DR. KEMPSON MADDOX, after thanking the speakers, said that with regard to Maclean's urea concentration test the physician was frequently left uncertain of its import, even when combined with an estimation of the blood urea, an invariable practice at Prince Alfred Hospital, but frequently neglected by private practitioners. Owing to its simplicity and correlation with clinical findings in thousands of cases this test would probably remain in general use, but its limitations must be thoroughly understood. Urea excretion depended on other factors than mere concentration by the kidney, for example, protein of the diet, rate of absorption from the intestines which depended on rate of emptying of the stomach, *et cetera*. These factors, however, if the urea was given fasting and a third specimen of urine examined, were not serious sources of fallacy. More important were the fairly common instances in which an apparently satisfactory concentration coincided with the clinical diagnosis of renal insufficiency or the urea concentration was at variance with the urea of the blood. In this regard, as with cardiac disease, it was imperative that a diagnosis and prognosis be finalized by every means possible. Contrary to Dr. Hansman's opinion, in the absence of obstruction to the urinary and intestinal tracts, that was, in the purely medical case, an estimation of the non-protein nitrogen was preferable to one of blood urea, in spite of the fact that a fume chamber was necessary and the end point broad, and in still anomalous cases one of blood creatinine, if raised, was more conclusive and alarming than either. Low blood creatinine, however, did not exclude a renal lesion. The "compensated kidney" described by Dr. Hansman was said to excrete equal and possibly satisfactory concentrations of urea in each specimen. This point could hardly meet with general acceptance, as it was impossible to distinguish how much reserve remained, and the supranormal figures which he quoted, at least in those cases with a low reading before the test, might equally well be interpreted as indicating a quicker and more complete response than usual, showing thereby a functional reserve which exceeded the ordinary bodily requirements. Some of the ancillary tests which should be used more frequently and in any case at all where any doubt remained, were: (i) The phenolsulphonephthalein test, especially where it was desired to distinguish between true nephritis and functional incapacity from chronic venous congestion of the kidney. This test was in far more general use in Europe than Australia and was held in high regard. (ii) The Mosenthal test was more frequently used in other cities of Australia and was an improvement on the simple Volhard water test. It was most useful in distinguishing true interstitial nephritis from secondary renal damage due to essential hypertension or arteriosclerosis. It was contraindicated in extra-renal œdema and acute nephritis. (iii) The diastase test had fallen into disrepute, but if combined with an estimation of that in the blood, it could well be retained because of its delicacy, as it might show impairment even after albumin and casts had returned to normal. (iv) Excretion of ten cubic centimetres of 10% solution of sodium thiosulphate given intravenously should reach approximately 30% in two hours.

In his opinion one of the most valuable and scientific confirmatory tests lay in the capacity of the kidney to excrete a measured dose of sodium chloride. According to Schlayer this and the phloridzin test were capable of distinguishing between the relative involvement of tubules and glomeruli, as both these substances were concerned chiefly with the tubules. If the tubules were healthy, excretion reached a normal maximum, favoured by glomerular diuresis. In early glomerular damage there was an exaggerated response as regards both salt and water. With greater damage excretion and output fell, associated with a rise in osmotic pressure of the blood. A rise in excretion with a fall in output indicated glomerular damage whereas an inadequate rise in excretion with a good output meant tubular damage. Alport's method was placing the patient on a diet of constant salt content and estimating the blood chloride and the percentage excretion for the previous night. At 8 a.m. the patient was given twelve grammes of sodium chloride in a pint of water. Urine was collected at hourly intervals until 1 o'clock, when thirty ounces of fluid were given, and urine was again collected at hourly intervals until 5 p.m. Normal salt concentration of the night urine usually varied from 0·6% to 0·8%. The maximum concentration in the morning urine should be from 1·35% to 1·8% chloride. The maximum concentration in the afternoon urine should be from 0·13% to 0·67%. The curve in chronic interstitial nephritis showed a poor excretion and concentration in all specimens. In chronic parenchymatous nephritis there might be no excretion of salt, as it was retained to compensate for the lowering of osmotic pressure in the blood through the loss of blood proteins. De Wesselow used a dose of four grammes and obtained concentrations of 0·8% to 0·9% chloride from normal persons, 0·2% in chronic interstitial nephritis and 0·1% in subacute parenchymatous nephritis.

When confronted with an albuminuria between 25 and 40 after thorough physical examination including ophthalmoscopy, Dr. Maddox would suggest the above tests to be performed *seriatim* until one was finally able to offer a prognosis. Using the results of two or more tests for

comparative purposes served to eliminate their individual disadvantages.

Once any suspicion of nephritis had been roused, the patient should be recalled for periodic retesting according to the test which has been found most sensitive in his case, and his vital *régime* regulated according to the findings from test to test. Mere repetition of blood urea examination was not sufficient.

With regard to children, a poor urea concentration, even less than 1%, should not be associated with quite the same expectation of an early lethal exit as with adults, as several cases had been reported in which such had existed for several years, and in one instance recovery had ultimately occurred. Similiarly, cardio-vascular hypertrophy in children had no constant relation to urea retention. The urea concentration and dye tests were the most practical for pædiatric use and associated with catheterization, and Dr. Maddox spoke of a device for the collection of urinary specimens from infants or young children.

The fear expressed by Dr. Holmes à Court that one would have to correct specific gravity according to albumin content could hardly apply to chronic interstitial nephritis, in which function tests were most used, because the faint and elusive quantity of albumin excreted did not usually reach a sufficient concentration to affect specific gravity.

Dr. Holmes à Court in reply said that considering the complexity of the issues involved there had been a striking uniformity of opinion in the three papers. He wished to establish the view that all the accepted tests were of relative value only; they did not and could not replace clinical observations in prognosis. There was sometimes a regrettable tendency to rely exclusively on laboratory tests in diagnosis. Even when Professor Maclean was in Sydney Dr. Holmes à Court had had an interesting experience with him in a case of chronic nephritis. The man had had a systolic blood pressure of 180 and a diastolic pressure of 130 millimetres of mercury as well as advanced albuminuric retinitis. The patient had been a very ill man and Dr. Holmes à Court had given a grave prognosis. Professor Maclean, relying on a blood urea content of fifty milligrammes *per centum* and on a moderately satisfactory result from a urea concentration test, had expressed the view that the patient might live two years. The patient had died within a few weeks. The clinical observations in this instance had proved of more prognostic value than the biochemical tests.

Dr. Hansman had carried out observations for him on one patient and had found a blood urea content of 53 milligrammes *per centum* and a blood creatinine value of nine. A bad prognosis had been given, but the patient had lived for three and a half years. It was necessary to evaluate tests of renal function in conjunction with clinical observation.

Dr. Earlam in reply said that he wished to thank Dr. Craig for amplifying several of the statements made in the course of the paper. As Dr. Craig stated, it was of primary importance in the surgical patient to correlate the functional findings with the patient's clinical condition.

Dr. Earlam also thanked Dr. Harris for his account of his experiences with the indigo-carmine test. Dr. Earlam had had small experience with it. What experience he had had placed him in agreement with Dr. Harris's remarks. It was an extremely sensitive test and he thought as a means of estimation of renal function, total or differential, was more or less on a par with the phenolsulphonephthalein. Which test was the better of the two was, he thought, rather a matter of personal opinion. Personally, he had been accustomed to employ the phenolsulphonephthalein test as a routine and principally in all probability for this reason he preferred it. He had been fortunate in escaping serious criticism, so concluded by thanking the other speakers for their appreciative remarks and the audience for their reception of the paper.

Dr. Hansman in his reply to Dr. Wardlaw said that he discussed the amount of water to be allowed with each individual patient and the amount was fixed according to the amount which the patient was accustomed to have. He always allowed some water up till twelve hours before, but none during the twelve hours immediately preceding the test. Dr. Wardlaw had referred to students whose urea concentration rose during the performance of the test and then remained constant. Dr. Hansman had stated in his paper that the type of patient he had described as having compensated kidneys was the type in which the concentration of urea in the first hour remained constant during the succeeding two hours.

In reply to Dr. Blackburn as to the importance of calculating the total quantity of urea excreted, Dr. Hansman said that Dr. Blackburn had been thinking of patients whose kidneys were impaired, and he required to know whether in a given time sufficient urea could be excreted to prevent accumulation of nitrogenous substances in the blood, whereas the type of patient with whom Dr. Hansman had been dealing, was the type which came for insurance examination, and it was important to determine whether their kidneys were unimpaired. In the tests it was the power to concentrate urea which was of importance, so that an excretion of 200 or 300 cubic centimetres of urine for three hours with a concentration of 1% was not the same as the excretion of a smaller amount of urine containing the same quantity of urea at a higher concentration. The body had to rid itself of urea, and so long as it could do so the kidneys might be considered good enough to see the patient through, but this was not the same as determining whether the kidney function was perfect.

In reply to Professor Mills who had said that Maclean considered 2% of urea in any hour to be satisfactory, Dr. Hansman said that Maclean was not here concerned with stating that the kidneys were perfect, but that a concentration of 2% denoted that the kidneys were not seriously damaged. As regards the accumulation of urea in patients with severe anæmia, his experience was not that of Professor Mills, and he recalled a recent case of a female, aged twenty-one, with an atypical pernicious anæmia whose red cell count was below 1,000,000 cells per cubic millimetre and in whom the blood urea was only 19 milligrammes *per centum*.

Dr. E. Shipton had raised the question of allowing patients food on the morning of the test. Dr. Hansman said that he had stressed the point that the urea concentration test was only a comparative test, so that if tests were to be of value the conditions under which they were carried out should be standardized. He pointed out that Harrison, whom Dr. Shipton stated was the authority on whom she allowed breakfast, held that if albumin were not present in the urine, there was no need to carry out tests of renal function, and he felt sure that everybody present had seen patients with renal damage in whom no albumin could be found with ordinary clinical tests. Dr. Hansman wished to stress once more the point that although the greatest concentration generally occurred in the third hourly specimen, this was not always so.

Dr. Tebbutt had raised the question of the ætiology of nephrosis. Recently some American workers had produced this condition for the first time experimentally. This was done by bleeding dogs, removing the red cells from the plasma and reinjecting the cellular elements.

POST-GRADUATE WORK.

THE following letter has been received from the Secretary of the Victorian Branch of the British Medical Association with a request from the Council that it be published.

British Medical Association House,
Tavistock Square,
London, W.C.1.
28th July, 1930.

Dear Sir,

The Council at its meeting on the 21st inst. had before it a report from the Science Committee relative to the resolution passed by your Branch urging the Association to create an organisation to provide full information and facilities for overseas graduates wishing to do postgraduate work in Great Britain and on the Continent.

I was directed to inform you that the Council received this resolution with much sympathy, but it

thinks you ought to know that for some years it has been doing a good deal of work in this connection. In this office we collect, publish and make available for the help of members of the Association information as to the main postgraduate facilities throughout the world. The editorial and medical departments, which are in touch with all the postgraduate organizations and centres at home, and with many of those abroad, co-operate in this work by means of the "Handbook for Recently Qualified Medical Practitioners," the Library, the Notice Boards in the House of the Association, and in response to individual applications. The current issue of the "Handbook for Recently Qualified Medical Practitioners" contains the following sections: "Postgraduate Study and Special Diplomas," "Specialisation," and "Some Fellowships, Scholarships, Studentships, Prizes and Research Grants open to Qualified Medical Practitioners." In addition we co-operate with the Fellowship of Medicine, which body has for many years now tried to do its best to organise postgraduate work in London, and I think I may say that in the very difficult circumstances it has done its work very well.

But the Association has always felt that some great effort was necessary, probably under the auspices of the Government, to establish in London a postgraduate school which would be a real credit to the Empire and would make available facilities which we know to exist. Partly in consequence of our efforts, the Ministry of Health appointed a Committee which some years ago recommended that it was essential that proper postgraduate facilities should be provided in London and that a hospital and medical school should be set aside for the purpose. At that time it was hoped that private donations would supply the need, but finding that this was not so, the Minister of Health appointed another Committee, and in April last it was reported that the Government had accepted the recommendation of the Committee that a British Postgraduate Hospital and Medical School should be established at the Hammersmith Hospital and that it was prepared to grant from public funds the sum of £250,000 for building and equipping the medical school and to make provision for grants through the University of London towards the maintenance of the school on the lines already applied to comparable institutions of University rank. On June 30th the Minister issued a statement showing the composition of a Committee which is to plan the construction of the School and lay down its constitution. The Committee is a very representative body, and includes several prominent representatives of the British Medical Association. The Council hopes, therefore, that before long London will be supplied with facilities which your Council has asked for, and you may be sure that no effort will be spared on the part of the British Medical Association to help the scheme forward.

In the meantime I hope your members will be informed of the work that the Association is already doing and that when they visit London they can obtain much useful information at this office, where we are always glad to see them.

Yours faithfully,

(*Signed*) ALFRED COX,
Medical Secretary.

Medical Societies.

THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN.

A MEETING OF THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN was held at the Hospital for Sick Children, Brisbane, on February 27, 1930, DR. G. P. DIXON in the chair.

Arthritis of the Wrist.

DR. J. S. AVERY showed a girl, aged nine years. On January 3, 1930, she had complained that a swelling on the back of the wrist had been noticed for two months; she did not know how it had commenced, but it had gradually swelled and had grown to its present size. A definite history of pain was not given.

On examination there had been a generalized swelling of the wrist, back and front, including the area over the bases of the metacarpal bones. The veins had been prominent. Movements had been restricted, there had been no dorsiflexion and very little palmar flexion; the fingers could be moved.

The heart sounds had been clear and the spleen was not palpable. There had been no other swellings and no glandular enlargement.

On January 14, 1930, the Wassermann test had yielded a "++++" reaction. Nothing abnormal had been detected on X ray examination. The wrist had been placed on a splint and the patient had been given "Kharsulphan" 0·15 gramme, inunctions of mercury three days a week and 0·48 gramme (eight grains) of iodide of potash three times a day.

On January 23, 1930, the condition had been the same and "Kharsulphan" 0·15 gramme had been given. The same treatment had been given on January 30, February 6 and February 12, 1930.

On February 20, 1930, thickening had still been present. There had been practically no dorsiflexion; other movements had been very restricted.

There was some redness on the palmar aspect and the wrist was more tender on handling than previously. Dr. Avery said that the diagnosis seemed to lie between congenital syphilis and tuberculosis. The former might cause an arthritis resembling that of tuberculosis, but it was often symmetrical, with no pain and very little impairment of movement. Regarding tuberculosis, Hale White stated that 40% of infections of joints occurred in the spine, 40% in the hip, 10% in the knee and the remaining 10% were distributed between the ankle, shoulder, elbow and wrist. There was a slow onset and it was often overlooked for three to four months; only one in twenty was recognized in the first month. At first there might be no pain and only slight impairment of movement, but these two things generally increased. Usually one joint only was affected.

The members present were of the opinion that antispecific treatment should be continued.

Spina Bifida.

DR. NEVILLE SUTTON showed a child, ten weeks old. The child had been born in December, 1929; he was a full-time child, the mother's fourth child and her only boy. The mother was forty years of age. She had had three girls and then one miscarriage and then this child.

The child had been born by the breech with bilateral *talipes equino-varus*, *spina bifida* and very open sutures of the skull.

The *spina bifida* had been of the common type, a meningo-myelocele, in the lumbo-sacral region. The skin had been deficient and a membranous area represented the pia-arachnoid and in the middle the thickened cord area had been attached to the sac. The area measured 8·75 by 7·5 centimetres (three and a half by three inches). It had been protected by spirit pads, but on one occasion there had been a slight leakage of cerebro-spinal fluid.

When the child was three weeks old Dr. Sutton had asked Dr. Dixon to see the child and with his assistance had operated; he had made an elliptical incision round the membranous area, had opened into the sac and had preserved the spinal cord area which was not adherent to the membrane. The child had been kept with the buttocks up.

Before operation the child had had incontinence of fæces and urine, which had been much improved since the operation. There had also been considerable spasticity, which had improved very much. There was still some spasticity of the flexors of the hip and also there was slight *genu recurvatum*. The double talipes had been

strapped straight away and the feet were now in aluminium splints and were much improved.

The cranial sutures were wide apart and the anterior fontanelle extended far back and the sagittal suture was wide. The normal size of the head for a new born infant was 33·75 to 35·0 centimetres (thirteen and a half to fourteen inches). In this child the measurement had been 36·75 centimetres (fifteen and a half inches) before operation. One week after operation it had been forty centimetres (sixteen inches). Ten weeks after operation it was 40·6 centimetres (sixteen and a quarter inches). The child held its head normally for its age.

Hydrocephalus.

DR. H. MATHEWSON showed a child that was suffering from hydrocephalus following meningitis. The child had had meningitis at three months of age in the Ipswich Hospital. At three years it had still been unable to walk. It was the only child, was full time; the mother had had no miscarriages. The child had been healthy at birth and had continued so till it was three months old. The child had been taken ill after measles, had been in hospital in a feverish condition, had not been unconscious, but had become stiff in the back. Lumbar puncture had been done frequently. The child had ultimately recovered after three months in hospital. It had been paralysed at the time, but the arms had recovered.

On leaving hospital the head had increased in size. Then, twelve months ago, it had been 55·6 centimetres (twenty-two and a quarter inches) in circumference and at the time of the meeting it was still the same. The muscles of the lower extremities were flabby and the child could not stand. The intelligence was moderately good and the child could speak. A Wassermann test was to be done.

A Case for Diagnosis.

Dr. Mathewson also showed a child, aged six months, for diagnosis. For the last three weeks the child had been irritable, crying a great deal and periodically dropping her head forward. This would happen three or four times a day, as though she had lost control. Three weeks previously, after a feed at six o'clock at night, the child when put down had screamed, the mother lifted her up, the head had dropped and then the child had lifted it again. She was slightly feverish and was not thriving. She had been holding her head up from three months like a normal child. The head and neck had been examined by X rays, but no abnormality had been discovered. The mother stated that one week before this condition was noticed the child had fallen out of bed, but nothing abnormal had been noted. The blood count did not indicate any inflammatory condition.

Tuberculosis of the Hip.

DR. A. V. MEEHAN showed a female child, aged eleven years, who was suffering from tuberculosis of the hip joint. As tuberculous joints were uncommon in practice in Brisbane, Dr. Meehan considered the patient was well worth showing. The condition was particularly interesting on account of the rapid progress of the disease. The child had been referred to him by Dr. G. P. Dixon.

The previous history was that the child had been admitted to hospital in October, 1929, with a history of pain in the left leg for a period of eight months. The pain had not been severe and there had been a slight limp. The condition had subsided and the child had been discharged. On December 21, 1929, the mother had noticed the limp again, the child had been unable to bend the left hip and had complained of a sensation of pins and needles in the left leg. On examination there had been a lump in the upper portion of the left thigh below the hip joint; the lump had been somewhat tender and had seemed to be intramuscular. It had been impossible to flex the left thigh.

An X ray examination in July, 1929, had revealed very little change, any change being mainly in the acetabulum. This was unusual and could easily have been passed over; there had been very little atrophy. On December 23, 1929, an X ray examination of the chest had revealed no abnormality. The skiagram of the left hip joint showed definite progress of the disease, still confined largely to the acetabulum. Early in January, 1930, the child had been admitted into hospital under Dr. Dixon and had later been referred to Dr. Meehan. On January 21, 1930, X ray examination had revealed definite changes in the head and in the acetabulum, there had been enlargement of the acetabulum, no outgrowth of bone, but absorption of bone. Shenton's line had been altered. About one month previous to the meeting the child had been put on an abduction frame; the lump had still been present and tender. It had been defined in the upper third of the thigh below the hip joint. The child had been febrile the whole time, the temperature varying daily from 37·2° to 38° C. (99° to 100·5° F.). The swelling had been needled and fluid obtained. This had been cultured and also injected into a guinea-pig. A film had been made of the fluid and examined and had been found to be teeming with tubercle bacilli. The results of the other tests had not yet been bacilli.

The only treatment was conservative treatment by extension on an abduction frame. The child was still running a febrile temperature at night and the prognosis was grave.

Tuberculous Meningitis.

DR. J. V. DUHIG showed two specimens of brains illustrating tuberculous meningitis. The first was particularly interesting, as the diagnosis had been made *ante mortem* by clinical and biochemical methods. The cerebro-spinal fluid from this patient had contained globulin and a lot of glucose. The second brain showed two huge tuberculomata, a rare condition.

Dr. Duhig also showed specimens of culture of tubercle bacilli by a new method on methyl violet with subsequent subcultures.

Abscess of the Iliac Glands.

DR. G. P. DIXON showed a child, aged eight years, with an iliac abscess and scoliosis. The child had been sent down from the north with a history of complaining of pain in the left hip region two days before. There had been marked scoliosis when the child stood up. The left leg was kept flexed and abducted and the hip joint had been diagnosed as tuberculous. Dr. Dixon had seen the child who had been admitted to hospital on February 3, 1930, with no further history.

On examination she had appeared to be a strong, healthy-looking girl who walked with a decided left-sided limp. When she lay down, the left limb had been flexed at hip and knee, there had been some shortening and wasting of the limb. There had been marked scoliosis and lordosis when the child attempted to extend the limb. The Wasserman test had yielded no reaction. X ray examination had revealed no abnormality. There had been an obvious swelling in the left iliac region—an abscess of the left iliac glands. This condition could be very misleading and on the right side was frequently mistaken for appendicitis.

When operating Dr. Dixon had used a grid-iron incision, had explored the retro-peritoneal space and opened into the glands and drained them. There was generally in these cases a history of sores on the feet; no explanation was forthcoming why the femoral glands should be missed and the infection go to the deeper iliac glands.

Syringomyelia.

DR. S. F. MCDONALD'S patient was shown by Dr. A. E. Patterson. The patient was a male child, aged eleven and a half years, who had complained of weakness and wasting of the right hand for eighteen months; it had come on slowly and was gradually progressing. Otherwise the child was well, except for septic sores over the arms and legs one month before the affection of the hand was noticed. The only history was that one year previously the child had burned his fingers with a match, but did not feel anything.

On examination there was definite involvement of the median and ulnar nerves of the right side; all the hand muscles supplied by these nerves were weak and wasted. The sensory fibres of the ulnar nerve were also involved, there was marked trophic disturbance of ring and little fingers and no response to touch, pin prick *et cetera*. The sensory fibres of the median nerve were affected to a less extent. The radial nerve appeared normal. Over the area supplied by the ulnar nerve there was no discrimination between heat and cold. The boy also stated that a few months previously he had also burned his hand on a kettle and did not know anything about it. There were no other muscles affected. The mother thought the left hand was beginning to get a little weak and that there was some wasting. There was no loss of sensation in the shoulder girdle or face and there was no wasting. The Wassermann test yielded no reaction. The trophic sore on the hand had been excised and the report was that "there was complete necrosis of all tissues and no resemblance whatever to leprosy" which, of course, came into the diagnosis. The diagnosis of syringomyelia was made.

Congenital Abnormality of the Anus.

DR. K. B. FRASER showed a child who was suffering from a congenital abnormality of the anus. The child had been born on the morning of December 29, 1929, when the nurse noticed something wrong with the anus. Dr. Fraser had seen the child that night and had had it admitted to the hospital and had operated next day.

He had found that there was complete absence of the anus, there was not even a dimple on the skin, due to the absence of the proctodeum. At operation a small rubber catheter had been passed into the bladder so that there would be no interference with the urethra, and then an incision had been made from the central point of the perineum to the coccyx. Dr. Fraser had extended the incision one inch inwards and had been unable to find the anus or any bulging when the patient coughed. He had then cut further back, had removed the coccyx, followed up the sacrum and finally found a bulge 3·75 centimetres (one and a half inches) higher up. This he had dissected out and opened, finding it to be the closed end of the rectum. He had finally got the edges of the rectum down and had made an artificial anus in the position of the coccyx. The child had got on well, had passed meconium. There were no muscle fibres in the position of the anus.

After a while urine had been passed through the artificial anus, presently it had been passed normally and then again through the anus, and this condition had been intermittent ever since. On the day of the meeting a small gum elastic catheter had been passed into the bladder and urine was passing out through it. Probably there was a very small recto-vesical fistula with a valve action, as Dr. Fraser was sure there had been no interference with the urethra. The child had been putting on weight satisfactorily.

Abscess of the Lung.

Dr. Fraser also showed a female patient, aged thirty-six years, who had been given a dental anæsthetic on May 8, 1929. When she was overhauled three years previously, her heart, lungs and blood pressure had all been normal. Under the anæsthetic twenty-six teeth were extracted, the patient lying flat on a table without a pillow; there had been no packing of the pharynx. Moderate ether anæsthesia induced by the open method had been used and the reflex had been present on completion of the extraction. The teeth had all been complete and had been counted by the dentist, his assistant and Dr. Fraser, both before and after the extraction, and no fragments of alveolar margin had been separated. Six days later, when Dr. Fraser was called to see the patient, she had complained of pain around the shoulder and a faint pleuritic rub had been detected. She had been sent to hospital immediately and an X ray examination had been made six days later. Clinically she had had diminished breath sounds in the left lung front and back, particularly over the upper lobe, and occasionally faint inspiratory crepitations had been audible below the left clavicle in front. The temperature had ranged from 37·8° to 38·9° C. (100° to 102° F.). The sputum had been free, but not copious, and had come up easily; it had the usual highly offensive odour.

No operative interference had been attempted, as the abscess was in the upper lobe. An X ray examination at this period had revealed an abscess cavity in the lower portion of the left upper lobe in the outer posterior aspect of the lung. It had been surrounded by an area of infiltration and had communicated with a dilated bronchus. No opaque foreign body had been seen. There had been a slightly diminished air entry into the lower lobe and diminished movement of the left dome and slight obliteration by the lateral sulcus.

Iodine and calcium had been administered freely and after three weeks the patient had begun to improve slowly, though the cough had lasted for several months and strength had been only very gradually regained. At the time of the meeting, nine months later, she was quite well and the lung picture was very different.

The X ray report on February 11, 1930, was that there was a band of fibrous tissue in the left upper lobe below the clavicle extending out to the periphery of the lung. This was all that remained of the former condition. No foreign body could be seen.

Dr. Fraser wished to stress five points with regard to administration of anæsthetics for dental purposes: (i) Preanæsthetic cleansing of the mouth, (ii) admission to hospital for the extraction, (iii) the position of the patient (the head over the end of the table), (iv) packing of the pharynx, or (v) use of intratracheal anæsthesia.

Post-Graduate Work.

COURSE ON OBSTETRICS IN MELBOURNE.

THE Melbourne Permanent Committee for Post-Graduate Work announces that the annual course in obstetrics will be held this year at the Women's Hospital, Melbourne, from November 10 to 24, inclusive. The course comprises attendance at the routine work of all departments of the hospital as well as special demonstrations and lectures by members of the honorary staff. Arrangements have been made whereby a limited number of graduates can reside at the hospital during the course. The fee for course is three guineas. An additional charge of four guineas is made for board at the hospital. Application should be made to the Honorary Secretary, 12, Collins Street, Melbourne.

NOTICE.

THE post-graduate demonstration which was to have been given by Dr. Howard Bullock at Sydney Hospital on September 27, 1930, has been postponed. An announcement of the date for the demonstration will be made in a subsequent issue.

Correspondence.

ACUTE OTITIS MEDIA.

SIR: Dr. Halford's criticism in the journal of this date of Dr. Godsall's article on acute *otitis media* of August 30 is unjustified. He states that the article is lacking in one very important particular, that is, details of the dressing and after-treatment of the meatus following paracentesis.

As this is one of a special series of articles dealing with diagnosis *per se*, Dr. Godsall was probably precluded on this account from mentioning details of treatment.

Dr. Halford is wise in drawing attention to the importance of preventing secondary infection of the discharge following paracentesis, as a certain number of cases do

go on into a chronic condition owing to a secondary infection occurring in the tympanic cavity, but only a small minority. To say, however, that "invariably failure to prevent this complication (that is, secondary infection) is the cause of the persistence of otorrhœa" and not necessarily because, as Dr. Godsall says, the mastoid is too deeply involved, is a statement to which the strongest exception must be taken. Its corollary is that every case of acute *otitis media* which does not clear up is due to failure to prevent a secondary infection of the tympanic cavity.

This is in direct opposition to the teaching and practice of modern otology, which regards (in the absence of a naso-pharyngeal infection) the persistence of an otorrhœa as due in the majority of cases to an infection of the mastoid antrum and cells.

The appropriate treatment is the early opening and draining of the mastoid process.

It is the failure in the past to recognize this source of infection that has led to the chronic running ear, with all its disabilities, which Dr. Godsall has so ably pictured at the commencement of his article.

Any statement, such as that of Dr. Halford's, which will withdraw attention from the mastoid antrum and may perhaps encourage practitioners "to go on with the drops," cannot be too strongly condemned.

Yours, etc.,

G. A. D. McARTHUR.

85, Spring Street,
Melbourne,
September 13, 1930.

PITUITARY GLAND PREPARATIONS.

SIR: In today's issue of your journal the following statement in the article from the pen of Dr. E. Sydney Morris occurs: "It may be mentioned that many practitioners complain of the inefficacy of certain pituitary gland preparations in controlling serious hæmorrhage . . ."

While agreeing with this, one would like to hear expressions of opinion regarding individual response to pituitary gland extracts. I have kept mainly to Parke, Davis preparations ("Pituitrin" and "Pitocin"), but have also tried three other brands.

Its administration in the second stage of labour sometimes acts most successfully, whereas in other cases it seems to make no appreciable difference whatsoever.

Is there any way to ascertain if this is only an apparent difference?

If, on the other hand, it be a real one, is it to be ascribed to the idiosyncrasy of the patient or to variation in standardization of the medicament?

Yours, etc.,

A. W. J. BULTEAU.

Pittwater Road,
Gladesville.
September 6, 1930.

THE EFFECTS OF GASSING.

SIR: I am trying to investigate the end results of gassing in ex-soldiers.

For this purpose I am writing to ask whether any medical men who suffered from gassing on service would be good enough to let me have the following particulars.

1. Age and present type of work, for example, general or special practice, laboratory work *et cetera*.
2. Date of gassing, place and nature of gas, for example, mustard gas, phosgene or mixtures.
3. Severity and length of time away from unit or whether was invalided to Australia.
4. Symptoms, signs and treatment.
5. Present condition, with special reference to (i) weight, (ii) cough and expectoration, (iii) temperature, (iv) dyspnœa and tachycardia, whether any difference, for example, is dyspnœa more prolonged than tachycardia, (v) symptoms and signs, (vi) an X ray report, if possible by a radiologist accustomed to the study of the "soldiers' lung."
6. General notes on mode of life—games, capacity for brain work and liability to infections.

The foregoing would be most helpful in the study of what is becoming an exceptionally difficult national problem.

Especially am I anxious to get into touch with a medical officer whom I saw in the Duchess of Westminster's Hospital at Le Touquet in May, 1918, after he had been badly gassed (mustard) at Villers Bretonneux.

Yours, etc.,

S. F. McDONALD, M.D., M.R.C.P.,
Physician Rosemount Repatriation Hospital.

"Craigston,"
217, Wickham Terrace,
Brisbane.
September 12, 1930.

MEDICAL ARTICLES IN THE DAILY PRESS.

SIR: The appearance in a recent number of a lay newspaper of an abstract of a contribution which appeared in THE MEDICAL JOURNAL OF AUSTRALIA and which at the same time gave in a headline the name of the author of the article in question, prompts me to make an explanation for the information of your readers.

THE MEDICAL JOURNAL OF AUSTRALIA is sent as an exchange to certain newspapers and in that way, of course, it comes to their notice.

These newspapers are being communicated with, requesting that when any abstracts are being made of contributions appearing in THE MEDICAL JOURNAL OF AUSTRALIA, the names of the authors be not disclosed.

I may add that the particular author referred to is at present in the United States of America.

Yours, etc.,

T. W. LIPSCOMB,
Chairman of Directors, Australasian Medical Publishing Company, Limited.

Sydney.
September 18, 1930.

WORKERS' COMPENSATION INSURANCE PRACTICE.

SIR: With reference to the correspondence *re* above in your journal of August 16 and September 6, both your correspondents refer to "medical referees" and, in doing so, display their ignorance of the Act they are criticizing. Under the *Workers' Compensation Act*, 1926, and its amendments the only medical referees recognized are those officially appointed by the Workers' Compensation Commission under the provisions of the Act. The Act specifically states that "where a medical referee has been employed as a medical practitioner in connexion with any case by or on behalf of an employer or worker or by any insurer interested he shall not act as a medical referee in that case." Medical referees are officers of the Commission while so acting and, as such, must be absolutely impartial in their certificates and written opinions, copies of which are supplied to both parties.

What your correspondents have confused with "medical referees" under the Act are the medical advisers employed by the insurance companies. Many insurance companies now have medical advisers attached to their staffs, recognizing the importance of early medical attention to injured workers and the necessity for advice to themselves on medical matters. The majority of the medical profession has adapted itself to the entirely new conditions brought about by the *Workers' Compensation Act* of 1926, but there are still a few who seem to be uncertain of their position.

With regard to disputed accounts for medical fees, the British Medical Association has an advisory committee and the medical officers attached to the staff of the Commission are always willing to assist in any conciliatory way.

The Workers' Compensation Commission alone can give any decision, which is final, as to the sum which an employer shall pay to an injured worker for medical and hospital treatment. But the Commission does not determine direct claims as between an insurance company and a medical practitioner. The Act does not vest a medical practitioner with a right to recover from an employer or his insurer the cost of treatment of an injured worker. The practitioner, of course, has his right at common law to recover such cost, in the usual manner, from the person who contracted for his service—the worker, employer or insurer, as the case may be.

Yours, etc.,

R. M. MACKAY,
Chief Medical Referee,
Workers' Compensation Commission.

Sydney.
September 12, 1930.

Obituary.

NEVILLE REGINALD HOWSE.

We regret to announce the death of Sir Neville Reginald Howse, which occurred in London on September 19, 1930.

Books Received.

THE PHYSIOLOGICAL PRINCIPLES OF HYDROLOGY, by R. G. Gordon, M.D., D.Sc., F.R.C.P., and F. G. Thomson, M.A., M.D., F.R.C.P.; 1930. London: Jonathan Cape; Sydney: Angus and Robertson. Crown 8vo., pp. 132. Price: 5s. net.

LIVER IN THE TREATMENT OF PERNICIOUS AND OTHER ANÆMIAS, by S. C. Dyke, D.M., M.R.C.P.; 1930. London: Jonathan Cape; Sydney: Angus and Robertson. Crown 8vo., pp. 128. Price: 5s. net.

Diary for the Month.

OCT. 1.—Victorian Branch, B.M.A.: Branch.
OCT. 2.—Federal Committee of the B.M.A. in Australia.
OCT. 2.—Queensland Branch, B.M.A.: Council.
OCT. 3.—New South Wales Branch, B.M.A.: Annual Meeting of Delegates of Local Associations with the Council.
OCT. 3.—Queensland Branch, B.M.A.: Branch.
OCT. 7.—New South Wales Branch, B.M.A.: Council.
OCT. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
OCT. 9.—New South Wales Branch, B.M.A.: Clinical Meeting.
OCT. 9.—Victorian Branch, B.M.A.: Council.
OCT. 10.—Queensland Branch, B.M.A.: Council.
OCT. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
OCT. 14.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
OCT. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 24.—Queensland Branch, B.M.A.: Council.
OCT. 28.—Queensland Branch, B.M.A.: Obstetrical Section.
OCT. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
OCT. 30.—New South Wales Branch, B.M.A.: Branch.
OCT. 30.—South Australian Branch, B.M.A.: Branch.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

CAIRNS HOSPITALS BOARD, QUEENSLAND: Medical Officer.
SWAN HILL DISTRICT HOSPITAL: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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